IN THE UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF DELAWARE

SMITH KLINE & FRENCH LABORATORIES LIMITED and SMITHKLINE BEECHAM CORPORATION d/b/a GLAXOSMITHKLINE, Plaintiffs,	)	Civil Action No. 05-197-GMS
v.	)	
TEVA PHARMACEUTICALS USA, INC., Defendant.	)	

PLAINTIFFS' PROPOSED FINDINGS OF FACT AND CONCLUSIONS OF LAW REGARDING INEQUITABLE CONDUCT

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Plaintiffs Smith Kline & French Laboratories Limited and SmithKline Beecham Corporation d/b/a GlaxoSmithKline (collectively "GSK") set forth below their proposed findings of fact and conclusions of law concerning Teva's allegation that U.S. Patent No. 4,824,860 ("the '860 patent") was procured by inequitable conduct and should be held unenforceable. In summary, defendant Teva Pharmaceuticals USA, Inc. ("Teva") has failed to carry its burden of proving by clear and convincing evidence that (1) anyone made any material misstatements or omissions during the prosecution of the '860 patent; (2) the alleged material misstatements and omissions were made with the intent to deceive the patent office; and (3) that, when balancing the alleged misrepresentations and alleged intent, that GSK's alleged conduct is so culpable that the patent should not be enforced.

PROPOSED FINDINGS OF FACT

I. INTRODUCTION

1. This is a patent infringement action in which GSK alleges that Teva's submission of an Abbreviated New Drug Application ("ANDA") seeking to market a generic version of GSK's REQUIP® (ropinirole hydrochloride)[1/] product for the treatment of Parkinson's Disease infringes claim 3 of the '860 patent. Teva concedes infringement but contends that the '860 patent is invalid (on the basis of anticipation and/or obviousness) and unenforceable for inequitable conduct.

2. After a three-day trial held on December 18, 2006 through December 20, 2006, the Court issued the following oral ruling:

> ORAL RULING (GENERAL VERDICT): FINDING THAT Teva has failed to prove by clear and convincing evidence: (1) that the prior art anticipates claim 3 of the '860 patent either expressly or

1/ Throughout this document, as was the case at trial, the terms "ropinirole" and "ropinirole hydrochloride" are used interchangeably.

> inherently; and (2) that the subject matter of claim 3 of the '860 patent would have been obvious to one of ordinary skill in the art at the time of the invention in view of the prior art references and knowledge in the field under the test set forth in *Graham v. John Deere*, 383 U.S. 1, 17 (1966). The Court shall reserve the specific articulation of its findings of fact and conclusions of law for a memorandum decision which shall be issued at a later date. The parties shall submit a briefing schedule regarding inequitable conduct and proposed findings of fact and conclusions of law.

Oral Ruling (December 20, 2006).

3. Pursuant to the Stipulation and Order Regarding the Post-Trial Briefing Schedule entered by the Court on January 17, 2007 [D.I. 167], as amended on January 31, 2007 [D.I. 171], GSK submitted its proposed findings of fact and conclusions of law relating to validity on February 7, 2007 ("GSK Proposed Validity Findings") [D.I. 172]. On that same day, Teva submitted [Proposed] Findings of Fact and Conclusions of Law on Teva Pharmaceuticals USA, Inc's Defense and Counterclaim of Inequitable Conduct ("Teva Proposed Inequitable Conduct Findings") [D.I. 184], and an accompanying brief ("Teva Inequitable Conduct Brief") [D.I. 185]. GSK is hereby responding to the Teva Proposed Inequitable Conduct Findings and is submitting contemporaneously a separate brief responding to the Teva Inequitable Conduct Brief.

4. GSK incorporates by reference herein the information set forth in paragraphs 1-22 of the GSK Proposed Validity Findings.

II. NO CLEAR AND CONVINCING EVIDENCE OF INEQUITABLE CONDUCT

A. Teva's Eleventh-Hour Allegations

5. Teva did not make any allegations of inequitable conduct until after the close of fact discovery in this case. *See* Teva's Motion for Leave to Amend Its Answer, Defenses, and Counterclaims, filed June 28, 2006 [D.I. 62], as corrected July 10, 2006 [D.I. 73]. The Court permitted Teva to amend its Answer and Counterclaims a month after fact discovery closed to add allegations of inequitable conduct with respect to the '860 patent and U.S. Patent No.

4,452,808 ("the '808 patent"). *See* Order Granting Motion to Amend/Correct, entered by the Court on July 28, 2006 [D.I. 87]. Notwithstanding the abandonment of its parallel validity and inequitable conduct challenges to the '808 patent, Stipulation to Partially Dismiss Claims Related to U.S. Patent No. 4,452,808 (Without Prejudice) entered December 14, 2006 [D.I. 162], and the general verdict the Court rendered against Teva with respect to its validity challenge to the '860 patent, Oral Ruling (December 20, 2006), Teva continues to pursue its inequitable conduct allegations concerning the '860 patent.

6. In attempting to support its inequitable conduct allegations with respect to the '860 patent, Teva relies almost exclusively on the deposition testimony of fact witnesses that the parties submitted to the Court by designation post-trial. GSK made available for deposition in the United States (and, in one instance, the United Kingdom) each of the fact witnesses whose deposition Teva sought, even though several of those witnesses are not currently (or never were) employees of GSK and all but one of the witnesses resides in the United Kingdom.

7. The witnesses whom GSK voluntarily produced for deposition included the following individuals:

- Dr. David Owen – a former GSK employee residing in the United Kingdom who was the Head of Pharmacology and is the sole named inventor on the '860 patent.
- Dr. Carol Harvey – a current GSK employee who served as the head of the ropinirole project team during the relevant time period.
- Roger Eden – a former GSK employee residing in the United Kingdom who served as a pharmacologist in one of Dr. Owen's laboratories during the relevant time period.

- Dr. Peter Giddings – a current GSK patent attorney residing in the United Kingdom who worked on the United Kingdom patent application that serves as the priority application for the '860 patent.
- Dr. Brenda Costall – an employee of the University of Bradford in the United Kingdom, who performed tests under contract for Dr. Owen and GSK to confirm Dr. Owen's hypothesis that ropinirole hydrochloride was a potential treatment for Parkinson's Disease.

8. While Teva claims ***for the first time*** in its ***post-trial*** filings that Annette Wright – a former GSK employee who served as a lab technician in Dr. Owen's lab – should have been named as an inventor on the '860 patent, Teva never even sought to take Ms. Wright's deposition. Nor did Teva seek during fact discovery to take the depositions of the Honorable Alan D. Lourie or Stuart R. Suter – the two former GSK employees whose names are listed as the patent attorneys on the face of the '860 patent, *see* '860 Patent (PTX 35)[2/] – or Vincent Fabiano, another former GSK employee whom GSK disclosed in its responses to Teva's interrogatories as a person involved in preparing the '860 patent application, *see* Teva Proposed Inequitable Conduct Findings ¶ 59 and DTX 140.

9. Teva seeks findings based on GSK's assertion of the attorney-client privilege and/or the absence of testimony from certain witnesses. However, at no point during this litigation did Teva move to compel or otherwise seek relief from the Court concerning access to GSK witnesses or any claim of privilege made by GSK.

10. Teva has alleged that the '860 patent was procured by inequitable conduct on the part of the named inventor, Dr. Owen, and unspecified GSK patent attorneys. In particular, Teva

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[2/] Copies of the trial exhibits have been submitted under separate cover.

claims that Dr. Owen and/or GSK's attorneys committed inequitable conduct by: (1) falsely declaring that Dr. Owen was the sole inventor of the subject matter claimed by the '860 patent; and (2) making certain alleged misstatements in the '860 patent specification concerning prior art dopamine agonist compounds. *See, e.g.,* Teva Proposed Inequitable Conduct Findings ¶¶ 8-12. For the reasons described in detail below, Teva has failed to prove by clear and convincing evidence that GSK's representatives made any material misstatements or omissions with the intent to deceive the United States Patent and Trademark Office ("PTO").

11. Teva concedes that it may not market its proposed ropinirole hydrochloride products until after the expiration of the '808 patent in December 2007. *See* Trial Tr. 72:16-73:3 (Jaskot).[3/] The '860 patent expires on May 19, 2008. *See* '860 Patent (PTX 35). Thus, by challenging the enforceability of the '860 patent, Teva seeks the ability to market its proposed products during the five-month period between expiration of the '808 patent and expiration of the '860 patent.

B. Allegations Concerning Inventorship

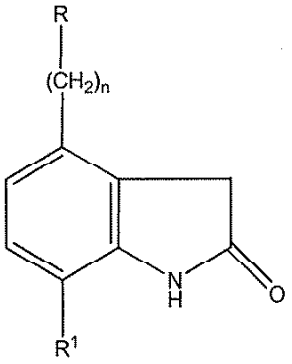
12. The record evidence demonstrates that Dr. David Owen was properly named as the sole inventor of the '860 patent and that GSK made no material misrepresentations concerning inventorship with an intent to deceive the PTO. Teva has failed to carry its burden of presenting clear and convincing evidence to the contrary.

3/ The abbreviation "Trial Tr." is used throughout this document to refer to the trial transcript, followed by the line and page numbers from the transcript and, where applicable, the name of the testifying witness in parentheses. Copies of the pages of the trial transcript that are cited in this memorandum are attached hereto as Exhibit A.

1. Prior GSK Patents Concerning Indolones

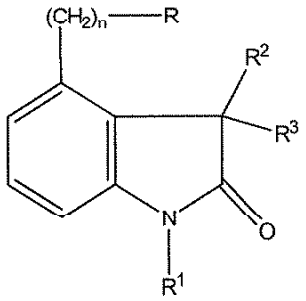
13. The '860 patent addresses the use of certain indolone compounds in the treatment of Parkinson's Disease. *See* '860 Patent (PTX 35). The '860 patent is one of a series of patents that GSK obtained relating to indolone compounds.

14. The first patent GSK obtained relating to indolone compounds was U.S. Patent No. 4,314,944 ("the '944 patent"), which issued on April 25, 1989, and names Dr. William Huffman and Dr. James Wilson as inventors. *See* '944 Patent (PTX 36). The '944 patent addresses "a new group of 4-aminoalkyl-7-hydroxy-2(3H) indolones which have a beneficial effect on abnormal conditions of the cardiovascular system." '944 Patent, Col. 1, ll. 4-7 (PTX 36). Claim 1 of the '944 patent is recited below:

> A compound of the structural formula:
>
> 
>
>
> in which R is amino, lower alkylamino, di-loweralkylamino; di-N-allylamino or N-allyl-N-lower alkylamino, R^1 is hydroxy or methoxy and n is an integer from 1-3; together with the pharmaceutically acceptable acid addition salts thereof.

15. On June 5, 1984, GSK obtained the '808 patent, which names Gregory Gallagher as the sole inventor. *See* '808 Patent (PTX 13). The '808 patent addresses a group of indolone compounds that lack the hydroxy group (i.e., an oxygen and a hydrogen) in the 7 position that

was featured in the '944 patent. *See* '808 Patent, Col. 1, ll. 9-47 (PTX 13). Claim 1 of the '808 patent is recited below:

> A compound of the structural formula:
>
> 
>
>
> in which: n is 1-3, R is amino, C_{1-6}-lower alkylamino, di-(C_{1-6}-lower alkyl)amino, allylamino, diallylamino, N-(C_{1-6}-lower alkyl)-N-allylamino, benzylamino, dibenzylamino, phenethylamino, diphenethylamino, 4-hydroxyphenethyl amino or di-(4-hydroxyphenethyl)amino, and R^1, R^2 and R^3 are, each, hydrogen or C_{1-4}-lower alkyl; or a pharmaceutically acceptable, acid addition salt thereof.

16. The '808 patent describes the claimed compounds as having activity at pre-synaptic peripheral D_2 receptors, *see id.* at Col. 4, ll. 31-44 (PTX 13); Trial Tr. 327:6-11 (Long); 556:15-557:16 (Jenner), and utility as dopamine agonists in treating disorders of the cardiovascular system, *see* '808 Patent, Col. 1, ll. 46-48; Col. 4, ll. 26-29 (PTX 13); *see also* GSK Proposed Validity Findings ¶¶ 77-81. The '808 patent does *not*: (a) make any mention of Parkinson's Disease; (b) state that ropinirole hydrochloride crosses the blood-brain barrier or has central nervous system activity; or (c) state that ropinirole has activity at post-synaptic dopamine receptors. *See* '808 patent (PTX 13); Trial Tr. 555:21-556:5; 559:7-15 (Jenner); 324:5-7; 300:18-24; 327:12-14 (Long).

17. One of the compounds claimed in the '808 patent is the compound known today as ropinirole hydrochloride. *See* '808 patent, Claim 5, Col. 10: 43-44 (PTX 13). GSK used the

internal designation SK&F 101468-A to refer to ropinirole during its development. *See* Deposition of Carol Harvey, May 25, 2006 ("Harvey Dep. Tr."), 16:2-8 (Exhibit B hereto).[4/]

2. Initial Development as a Cardiovascular Drug

18. In the years after its discovery, GSK scientists attempted to develop ropinirole as a cardiovascular drug. *See* Transcript of the Deposition of David Owen, May 26, 2006 ("Owen Dep. Tr."), 38:1-39:4 (Exhibit C hereto);[5/] Harvey Dep. Tr. 45:5-7 (Ex. B).

19. In the fall of 1985, development of ropinirole was transferred from GSK's operations in the United States to its facilities in Welwyn, England ("Welwyn"). *See* Harvey Dep. Tr. 42:2-43:10 (Ex. B). At that time, Welwyn had spare capacity to continue the necessary pre-clinical development work for the compound. *See id.*

20. At the time of the transfer, ropinirole was designated for development as a cardiovascular drug. *See* Harvey Dep. Tr. 45:5-7 (Ex. B). The understanding within GSK at the time was that ropinirole did not have central nervous system ("CNS") effects. *See* Harvey Dep. Tr. 49:7-51:1 (Ex. B); Owen Dep. Tr. 54:7-60:13 (Ex. C). Indeed, a 1985 publication by Gregory Gallagher (the named inventor of the '808 patent) and others describes a number of tests conducted on ropinirole and concludes: "These results indicate that 1(c) [ropinirole] *does not produce the central behavioral effects often seen with dopamine agonists*." Gallagher, Jr., G., et al., *4-[2-(Di-n-propylamino)ethyl]-2(3H)-indolone: A Prejunctional Dopamine Receptor Agonist,* J. Med. Chem., 28, 1533, 1535 (1985) ("the 1985 Gallagher article") (PTX 17) (emphasis added).

4/ A copy of the relevant pages of Dr. Harvey's transcript is attached hereto as Exhibit B.

5/ A copy of the relevant pages of Dr. Owen's transcript is attached hereto as Exhibit C.

3. Discovery of Anti-Parkinson's Potential

21. Dr. David Owen first became directly involved with the development of ropinirole when the compound was transferred to Welwyn. *See* Owen Dep. Tr. 42:19-43:2 (Ex. C). At the time, Dr. Owen was the senior pharmacologist for GSK in the United Kingdon and was responsible for a significant number of laboratories. *See* Owen Dep. Tr. 43:12-45:17 (Ex. C). Dr. Owen was trained in cardiovascular pharmacology. *See* Owen Dep. Tr. 244:20-246:15 (Ex. C). The laboratories under Dr. Owen's direction were focused on cardiovascular research. *See* Owen Dep. Tr. 54:22-55:8 (Ex. C).

22. Upon transfer of ropinirole to Welwyn, Dr. Owen determined that it was necessary to conduct some additional studies for the purpose of understanding the compound. *See* Owen Dep. Tr. 38:1-39:4 (Ex. C). More specifically, Dr. Owen decided that it was appropriate to conduct conscious animal cardiovascular measurements of heart rate and blood pressure. *See* Owen Dep. Tr. 43:12-45:17 (Ex. C). Those experiments were subsequently performed under Dr. Owen's direction. *See* Owen Dep. Tr. 43:12-45:17; 56:11-15 (Ex. C) ("So we undertook cardiovascular studies, that was to say cardiovascular studies were undertaken by people who reported to me, in labs for which I was responsible, cardiovascular studies in conscious animals.").

23. The individual who performed the experiments, under Dr. Owen's instruction, was Annette Wright. *See* Owen Dep. Tr. 43:12-45:17; 55:14-58:4 (Ex. C). Ms. Wright was a GSK lab technician who worked in one of Dr. Owen's laboratories under the immediate supervision of Roger Eden, a pharmacologist who reported to Dr. Owen. *See id*; Harvey Dep. Tr. 49:7-19 (Ex. B); Transcript of the Deposition of Roger Eden, April 27, 2006 ("Eden Dep.

Tr."), 42:7-22 (Exhibit D attached hereto).[6] The specific experiments Ms. Wright conducted involved the measurement of blood pressure and heart rate in spontaneously hypertensive rats. *See* Eden Dep. Tr. 70:20-71:12 (Ex. D). The experiments were performed in February 1986 and documented in Ms. Wright's laboratory notebook. *See* Laboratory Notebook of Annette Wright at GSK-REQ000385-86; 390-91; 395-96 (DTX 24); Eden Dep. Tr. 70:22-72:19 (Ex. D).

24. In the course of performing the experiments, Ms. Wright observed that there were some behavioral changes in the rats with which she was not normally familiar. *See* Owen Dep. Tr. 57:6-58:4 (Ex. C). She notified Dr. Owen of the changes, and he went to observe the rats at her request. *See id.* The specific behavioral effects that Ms. Wright and Dr. Owen observed included agitation and "stereotypy" (i.e., sniffing behavior). *See* Owen Dep. Tr. 55:9-58:4 (Ex. C); Laboratory Notebook of Annette Wright at GSK-REQ000385-86; 390-91; 395-96 (DTX 24).

25. Dr. Owen's interpretation of this behavior was that ropinirole had CNS effects. *See* Owen Dep. Tr. 55:9-58:4 (Ex. C).[7] His first reaction upon drawing this conclusion was "oh dear, there's a problem here," Owen Dep. Tr. 58:8-9 (Ex. C), because CNS effects were undesirable, as a general rule, in compounds targeted for cardiovascular development, *see* Owen Dep. Tr. 55:9-58:4 (Ex. C).

26. After his initial reaction of concern, however, Dr. Owen began to see the CNS effects of ropinirole as an advantage and came up with the idea that ropinirole could be useful in treating Parkinson's Disease. In Dr. Owen's own words:

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[6] A copy of the relevant pages of Mr. Eden's transcript is attached hereto as Exhibit D.

[7] Teva relies on Dr. Harvey's testimony to suggest that "anyone who ran tests in [a certain] dosage range saw the central effects of the drug," *see* Teva Proposed Inequitable Conduct Findings ¶ 20. That testimony, however, addresses a report distributed in March 1986 –*after* the experiments in which Ms. Wright and Dr. Owen observed the behavioral effects in rats that led Dr. Owen to conclude that ropinirole had CNS effects. *See* Laboratory Notebook of Annette Wright, GSK-REQ000385-86; 390-91; 395-96 (DTX 24); *see* Harvey Dep. Tr.141:10-146:16 (Ex. B); DTX 71.

> I formed the view that actually, this may actually be an advantage, rather than a problem, and that we may have a drug which would be useful for the treatment of Parkinson's disease. So over a short period of time, and I can't be precise what I mean by that, but not very long, but definitely not instantaneously, I conceived the hypothesis that this may be a treatment for Parkinson's disease.

Owen Dep. Tr. 58:13-21 (Ex. C).

27. Dr. Owen's observation of stereotypy and agitation in rats was "a factor, beyond a doubt, probably a rather large factor" in forming his hypothesis. *See* Owen Dep. Tr. 60:1-13; 58:22-59:5 (Ex. C).

28. To the best of Dr. Owen's recollection, the first time he shared his hypothesis with anyone was in a telephone conversation with Dr. Brenda Costall at the University of Bradford ("Bradford") in the United Kingdom. *See* Owen Dep. Tr. 63:10-22 (Ex. C).

29. Before contacting Dr. Costall, Dr. Owen knew that she and her colleagues had published extensively in CNS pharmacology. *See* Owen Dep. Tr. 96:11-97:7 (Ex. C). He also knew that Dr. Costall had developed animal models for studying anti-Parkinson's activities. *See* Owen Tr. 70:15-71:5; 112:13-113:1 (Ex. C).

30. Dr. Owen asked Dr. Costall and her colleagues to test his hypothesis that ropinirole could be used to treat Parkinson's Disease and also to put it into context by determining whether the compound had any other effects on the central nervous system. *See* Owen Dep. Tr. 109:3-14; 104:18-106:1; 169:9-21 (Ex. C).[8/]

31. Before Dr. Owen contacted Dr. Costall, he had a definite and permanent idea in his mind that ropinirole could be used to treat Parkinson's disease. *See* Owen Dep. Tr. 72:3-13

[8/] Teva's attempt to rely on GSK meeting minutes to suggest that Bradford was asked to evaluate the CNS effects of ropinirole and independently came up with the idea of its anti-Parkinson's potential, *see* Teva Proposed Inequitable Conduct Findings ¶¶ 23-24, is belied by the record. Both Dr. Owen and Dr. Costall testified that Dr. Owen first brought up the idea of using ropinirole to treat Parkinson's Disease, *see* Owen Dep. Tr. 72:3–13 (Ex. C); Deposition of Brenda Costall, May 4, 2006 ("Costall Dep. Tr."), 57:10-22 (Exhibit E attached hereto).

(Ex. C). Dr. Owen testified that, prior to his approaching Dr. Costall, "it was absolutely in my mind that it could be a treatment for Parkinson's disease, that is a hypothesis that was formed, but I needed others to confirm it, I didn't have the experimental models under my direction to confirm that. I wanted people who if they confirmed it, others would believe them." *Id.*

32. Dr. Carol Harvey was the global project team leader for ropinirole at GSK from late 1985 until some point in 1988. *See* Harvey Dep. Tr. 16:12-16 (Ex. B). Dr. Harvey had contemporaneous conversations with Dr. Owen that corroborate his conception of the idea of using ropinirole to treat Parkinson's Disease before he contacted the external investigators at Bradford:

> Q: And was it a result of the in-house [studies] that there was a determination to go to the external investigators?
>
> A: Yes.
>
> Q: Who made that determination?
>
> A: That was Dr. David Owen.
>
> Q: And why did he do that?
>
> A: He felt that the compound might have potential in other areas and should be more fully evaluated.
>
> Q: Potential in what areas?
>
> A: Specifically in Parkinson's disease.
>
> Q: Why did he think that?
>
> A: From his observations in the animals and his knowledge of the area.
>
> Q: And what's your basis of that understanding?
>
> A: My conversations with him.

Harvey Dep. Tr. 62:5-22 (Ex. B).

33. Dr. Costall's deposition testimony also confirms that David Owen first came up with the idea of using ropinirole to treat Parkinson's Disease. She acknowledged that the work that she and her Bradford colleagues performed involved "confirm[ing] the hypothesis *that had*

been formulated at Smith Kline & French that ropinirole may have anti-Parkinson potential." Costall Dep. Tr. 32:11-19. Dr. Costall confirmed that Dr. Owen initiated contact with her, informed her of his hypothesis that ropinirole had anti-Parkinson potential, and asked her to collaborate in confirming that hypothesis:

> Q: Let me ask you, how did you first get involved in doing this research work on ropinirole for Smith Kline?
>
> A: I received a telephone call from Dr. David Owen from Smith Kline & French.
>
> Q: And what did you and Dr. Owen discuss?
>
> A: Dr. Owen described to me some behavioral effects that had occurred whilst observing this compound then known as 101468, *and he said that in his view, that this represented a compound with anti-Parkinson potential*, and asked whether we would be prepared to collaborate with them in confirming his hypothesis.
>
> Q: Can you tell me approximately when that conversation was?
>
> A: Approximately 1986.

Costall Dep. Tr. 57:10-58:3 (Ex. E) (emphasis added).

34. In addition to describing his hypothesis, Dr. Owen explained to Dr. Costall during that initial call that he had seen certain behavioral effects during the testing of ropinirole. *See* Costall Dep. Tr. 57:10-58:3 (Ex. E). He specifically mentioned stereotyped behavior (or stereotypy), "which is a very specific indication in animals on the effect of the D2 receptors and that would indicate anti-Parkinson potential." Costall Dep. Tr. 69:8-21 (Ex. E). Dr. Owen explained to Dr. Costall that he wanted Bradford to conduct an investigation to confirm his hypothesis because GSK did not have the facilities (including animals and experience in behavioral testing) in-house. *See* Costall Dep. Tr. 73:11-75:3 (Ex. E).

35. Dr. Costall testified that there were at least two contracts between GSK and Bradford relating to the work Bradford performed on ropinirole. *See id.* 126:13-127:7 (Ex. E).[9/] Pursuant to this contractual relationship, Bradford was compensated for the costs of the animals and for the personnel involved in carrying out the work. *See id.* 96:10-97:1 (Ex. E). Moreover, Bradford was contractually required to assign to GSK any intellectual property it developed, which was consistent with Bradford's standard practice in dealing with pharmaceutical companies. *See id.* 88:13-90:2 (Ex. E). The Bradford researchers were also required to turn over the results of their work to GSK and were prohibited from publishing information about that work without GSK's approval. *See* Eden Dep. Tr.108:10-18 (Ex. D).

36. The tests used by Dr. Costall in evaluating ropinirole at the request of Dr. Owen and GSK were the same tests she had used previously in performing anti-Parkinson's research on other compounds. *See* Costall Dep. Tr. 80:2-10 (Ex. E). The research Dr. Costall and her team performed on ropinirole and the conclusions they reached were done in consultation with GSK. *See id.* 312:9-313:11 (Ex. E).

37. The work carried out by Bradford "confirmed the hypothesis presented by Dr. Owen that ropinirole had anti-Parkinson's potential." *Id.* 87:1-6 (Ex. E).

38. In 1986 or 1987, GSK's Preregistrational Affairs Department issued a report (SK&F Report No. PW005BA) documenting Bradford's initial round of studies and conclusions concerning ropinirole. *See* DTX 35.[10/] The report notes on its face that it was "prepared for Dr. D.A.A. Owen" by "B. Costall and R.J. Naylor" of Bradford. *Id.* at GSK-REQ001028. It

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9/ While Dr. Costall was not able to locate and produce any contracts between Bradford and GSK because they had been destroyed with Bradford's archives, she testified unequivocally about the existence of a contractual relationship and its general terms. *See* Costall Dep. Tr. 54:20-57:9; 88:13-91:8; 96:10-97:1; 126:13-127:7 (Ex. E).

10/ The cover page of the report contains two different dates: September 1986 and July 9, 1987. *See* DTX 35 at GSK-REQ001028.

summarizes the experimental work that Bradford performed at Dr. Owen's request and confirms the hypothesis he previously shared with Dr. Costall concerning ropinirole's anti-Parkinson potential. *See* DTX 35 at GSK-REQ001062 ("SK&F 101468A thus presents a novel dopamine agonist having antiparkinson, antidepressant and anxiolytic potential.").

39. Contrary to Teva's suggestion, *see* Teva Proposed Inequitable Conduct Findings ¶ 30, the results in SK&F Report No. PW005BA do not contradict the observation of stereotyped sniffing behavior that contributed to Dr. Owen's hypothesis concerning ropinirole's anti-Parkinson's potential. The experiment at issue, which is described in Part 4 of the report, provides a scale from 0 through 4 for measuring stereotyped behavior, with 0 representing no stereotypy, and 1 through 4 representing progressively more intense levels of stereotypy. *See* DTX 35 at GSK-REQ001042-1045. A graph illustrating the experimental results shows that ropinirole induced level 1 stereotyped behavior (period sniffing, head or limb movements) at all administered doses. *See id.* at GSK-REQ001045. This is perfectly consistent with the "classic stereotyped sniffing" behavior observed by Dr. Owen and reflected in Ms. Wright's lab notebook. *See* Laboratory Notebook of Annette Wright at GSK-REQ000385; GSK-REQ000391 (DTX 24).

40. There is no factual basis for Teva's suggestion that SKF Report No. PW005BA is evidence of invalidity under 35 U.S.C. § 102(f).[11/] First, it is evident from the face of this document that it is an internal GSK document issued by GSK's Preregistrational Affairs Department. Second, although the document reflects work performed by Dr. Costall and her colleagues under contract with GSK, Dr. Costall made quite clear in her testimony that Bradford's work was undertaken at the request of Dr. Owen based on Dr. Owen's prior

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11/ 35 U.S.C. §102(f) provides that a person shall be entitled to a patent unless "he did not himself invent the subject matter sought to be patented."

conception and performed in active consultation with GSK. *See* Costall Dep. Tr. 312:1-313:11 (Ex. E).

41. Bradford subsequently performed additional studies on ropinirole that substantiated its earlier studies and provided further confirmation of the compound's anti-Parkinson potential. *See* Harvey Dep. Tr. 79:3-80:9 (Ex. B).

4. The Application Leading to the '860 Patent

42. The priority application for the '860 patent (United Kingdom 8712073) was filed on May 21, 1987, and the U.S. patent application was filed on May 19, 1988. *See* '860 Patent (PTX 35). David Owen is named as the sole inventor in the '860 patent application, as well as on the patent. *See* DTX 19 at GSK-REQ000539-554; '860 Patent (PTX 35). The claims in the '860 patent are substantially the same as those in the '860 patent application. *See id.*

43. The '860 patent lists two individuals as GSK's patent agents or attorneys: Stuart R. Suter and Alan D. Lourie. *See* '860 Patent (PTX 35). Neither of these individuals is currently employed by GSK, nor did Teva seek to depose them in this action.

44. Peter Giddings is currently the Head of Patent Administration and Information in GSK's Corporate Intellectual Property Department. *See* Transcript of the Deposition of Peter Giddings, July 20, 2006 ("Giddings Dep. Tr."), 9:5-10:6 (Exhibit F hereto).[12/] Dr. Giddings is a U.K. Chartered Patent Agent and a European Patent Attorney, *see* Giddings Dep. Tr. 12:2-13 (Ex. F), and he is the only patent attorney currently employed by GSK who had any role in the application leading to the '860 patent. Specifically, Dr. Giddings was responsible for drafting the U.K. priority application on which the United States application is based. *See id.* 17:6-16 (Ex. F). Dr. Giddings testified that the prosecution of the United States '860 patent application

12/ A copy of the relevant pages Dr. Giddings' transcript is attached hereto as Exhibit F.

was primarily the responsibility of the attorneys in GSK's United States offices. *See id.* 103:21-104:6 (Ex. F).

45. Dr. Giddings testified that he does not remember particular details concerning the preparation of the applications leading to the '860 patent (which occurred almost twenty years ago), including the investigation of inventorship and which documents Dr. Owen reviewed prior to the filing of the U.K. or U.S. patent applications. *See* Giddings Dep. Tr. 47:1-14; 99:21-100:16; 101:11-102:12 (Ex. F).

46. Dr. Giddings did, however, testify about the general practice he followed at the time with respect to the preparation of patent applications:

> Q: Can you describe for me your typical process for drafting a patent application?
>
> MS. WIGMORE: Again, you may testify about your general practice, but you may not in the course of your answer reveal the substance of any attorney-client communications.
>
> THE WITNESS: Yes, from a general practice, perspective, usually the first point would be somebody from R&D would come to me with some information that they would like to discuss whether or not that's patent -- it is patentable, and we would look at what information they have and discuss -- discuss putting together a claim, a patent claim for the invention bearing in mind any prior art that may be out there affecting the invention and also looking at what might be a reasonable prediction for a generic claim based on the information that we have.
>
> And from the claim, my practice was then to turn to drafting a description of the patent application, which is basically the description of how to make and use the claimed invention; and at that point I would need from the usually scientists detailed information as to the specific examples of the application, data and so on.
>
> And then I would arrive at a draft patent application, and then I would have the people in R&D who have provided me the information review the application and be comfortable that everything that I've said is correct and complete.

Giddings Dep. Tr. 33:2-34:9 (Ex. F).

47. Dr. Giddings also described his usual practices when making inventorship determinations:

> Q: What type of investigation would you do to determine the proper inventorship of an application?
>
> MS. WIGMORE: And you may answer based on your general custom and practice. You may not in the course of your answer reveal the substance of any attorney-client communications to the extent you remember any.
>
> THE WITNESS: As a general practice, I would talk to the people who provided me the information relating to the invention in the first instance and I would ask them to provide me with details of what they thought their involvement comprised and then also ask them to tell me of any other people who were involved with the work, as well; and then I would talk to those people to see -- to get their view, as well, and from that information I'd make a designation of inventorship.

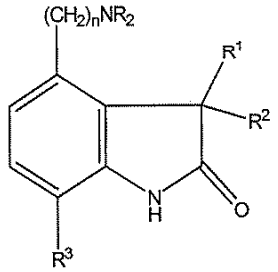
Giddings Dep. Tr. 29:17-30:11 (Ex. F). He further testified that he had no reason to believe he had departed from that practice with respect to the application that ultimately became the '860 patent. *See id.* 47:1-14 (Ex. F).

48. Dr. Owen testified that the '860 patent application was prepared by the GSK patent department "according to their own professional expertise after discussions with me and no doubt with others." Owen Dep. Tr. 25:21-27:6; *see also id.* 131:13-132:12 (Ex. C). In Dr. Owen's words: "I disclosed information to the patent department and, in my view, quite correctly, the patent department were the determinants of who was the inventor." Owen Dep. Tr. 80:21-81:9 (Ex. C). He further explained that he simply does not remember any specific documents or information he gave to the GSK patent department concerning his invention or the preparation of the U.K. patent application from which the '860 patent claims priority. *See id.* 94:9-95:7; 208:4-21 (Ex. C).

5. The Genus Claim

a) Drafting of Genus Claims Generally

49. Claim 1 of the '860 patent (which is not being asserted by GSK in this action) was substantially unchanged from the version of the claim that appeared in the '860 patent application. *Compare* '860 patent, Col. 6, l. 67-Col. 8, l. 4 (PTX 35) with DTX 19, at TEV-RQEXP000554. Claim 1 of the '860 patent is recited below:

> A method of treatment of Parkinson's disease which comprises administering an effective non-toxic amount for the treatment of Parkinson's disease of a compound of the following structure:
>
> 
>
>
> in which each group R is hydrogen or C1-4 alkyl; R1 and R2 are each hydrogen or C1-4 alkyl; R3 is hydrogen or hydroxy; and n is 1 to 3; or a pharmaceutically acceptable salt thereof to a subject in need thereof.

50. Claim 1 of the '860 patent is a "generic" or "genus" claim, *see* Giddings Dep. Tr. 37:8-12 (Ex. F), that encompasses a number of indolone compounds.[13/] A genus claim can, as in this case, take the form of a structural formula that captures the idea embodied in the specific example or examples provided by the inventor. As GSK's in-house patent attorney, Dr. Giddings, testified:

13/ While Teva repeatedly claims that claim 1 of the '860 patent encompasses "at least 750 compounds," *see, e.g.*, Teva Proposed Inequitable Conduct Findings ¶ 33, Teva offers no record support for that assertion.

> Q: Can you tell me what you mean by 'a generic claim'?
>
> A: Yes. Normally when the scientists come to us they have a specific example or specific examples of work, and it is my job as a patent attorney to look at what they have and see if we can -- see if from that we can create a general -- a general formula that covers -- that covers the really the invention that they have so there may be a number -- so they have a number of individual examples within that can be encompassed within a broad -- sorry, not necessarily a broad but in the generic formula that protects really the invention, if you like, or the idea that they have which is embodied in those individual examples.

Giddings Dep. Tr. 35:22-36:14 (Ex. F).

51. Dr. Giddings made clear in his testimony that the idea behind a genus claim is not to broaden the invention but, rather, to encompass the concept of the invention:

> Q: Is the generic claim typically broader than the idea that the person from R&D who initially approaches you has come up with?
>
> MS. WIGMORE: Object to the form of the question. I also instruct you not to reveal the substance of any attorney-client communications in your answer, but you can answer.
>
> THE WITNESS: I don't think it's – it's not correct to say that it's broader. It necessarily encompasses more than the specific examples that you have been given but its intent is to cover the concept of the invention, which, rather than saying it's broader than the invention -- or it's not correct to say it's broader than the invention. The invention is the generic claim, and it's defined and it's supported by the specific embodiment -- the specific examples that you have. So it's -- it's -- it's often not -- to say it's one invention, it's not broadening the invention.

Giddings Dep. Tr. 39:5-40:1 (Ex. F).

52. Although Dr. Giddings did not remember how the genus claim in claim 1 of the '860 patent was arrived at, *see* Giddings Dep. Tr. 38:9-17 (Ex. F), he testified about his typical practice for drafting genus claims:

> Q: So under the typical process, that generic claim would be something that you came up with; is that correct?
>
> MS. WIGMORE: And again, in your answer you may reveal – you may discuss your typical practice, but if you have any recollection of the specific process by which you drafted Claim 1, I

> instruct you not to reveal the substance of any attorney-client communications about that.
>
> THE WITNESS: With respect to this specific claim, I don't recall how I have that claim – how that claim was arrived at.
>
> As a matter of general practice, it's something I do in discussion with the people involved with the invention. So it's a it's a joint discussion about the generic scope.

Giddings Dep. Tr. 37:14-38:7 (Ex. F).

53. The process for drafting genus claims described by Dr. Giddings is consistent with industry practice, as reflected in patent law treatises and handbooks on patent drafting. *See, e.g.*, Robert C. Faber, *Landis on Mechanics of Patent Claim Drafting* § 10:1.1 (5th ed. 2005) ("Broad coverage means not only that every particular preferred disclosed embodiment is protected in the claims, but that the claims cover all expected and unanticipated equivalents that competitors and others may later develop and all intentional and unintentional copies of the claimed invention which embody the inventor's concept . . . . It is the claim drafter's job to have written the claims in the application to not only cover what the attorney and the inventor/client could at the time of application prosecution have envisioned as competing products, but to cover competitive products which neither the inventor nor the attorney thought of or could even have imagined at the time, but which employ the concept of the invention."); Jeffrey G. Sheldon, *How to Write a Patent Application*, Practicing Law Institute § 6.5.3 (2006) ("The broadest claim should be as broad as possible in view of the prior art. As long as the broad claim is not anticipated by art known to the inventor, it cannot hurt to ask for the broad claim. At worst, the examiner will not allow the broadest claims. Thus, it is recommended that the practitioner be greedy when initially writing the application."); Irving Kayton, *Kayton on Patents* 3-1, (2d ed. 1983) ("During the prosecution stage the drafter will naturally attempt to write one claim that is

as broad as the prior art of which he is aware will permit and that is supported by the disclosure in his patent application.").

54. Dr. Giddings testified that he does not believe he was an inventor of the '860 genus claim:

> Q: So based on your recollection sitting here today, you can't say that you were not the inventor of the generic claim that is Claim 1 of the '860 patent, correct?
>
> MS. WIGMORE: Objection. And I instruct you not to reveal the substance of any attorney-client communications in your answer if you recall any.
>
> THE WITNESS: I have no belief that I was the inventor of that claim.

Giddings Dep. Tr. 45:10-19 (Ex. F).

55. All of the examples in the '860 patent relate to ropinirole hydrochloride, and not the other compounds encompassed by claim 1. *See* '860 Patent, Col. 3, l. 40-Col. 6. l. 65 (PTX 35).[14/] The Examiner allowed claim 1 as an appropriate generic claim based on the testing shown for ropinirole.

b) Reasonableness of the Genus

56. As noted above, before filing the '860 patent application, GSK had obtained other patents relating to indolone compounds, including the '944 patent (PTX 36) and the '808 patent (PTX 13). *See supra* paragraphs 14-15. Both of those patents contain genus claims. *See id.* Dr. Paul Bartlett, a medicinal chemist who testified as an expert on GSK's behalf at trial, compared the genus claim in claim 1 of the '860 patent to those in the previously issued '944 and '808

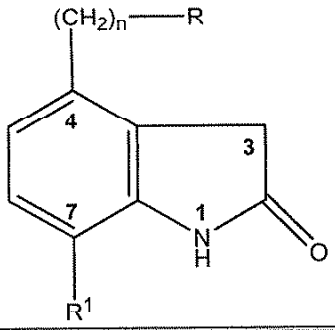
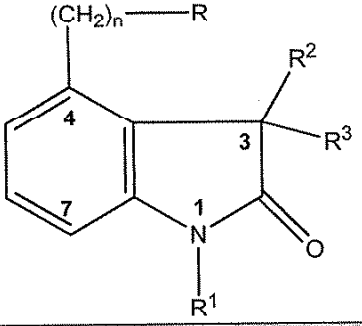
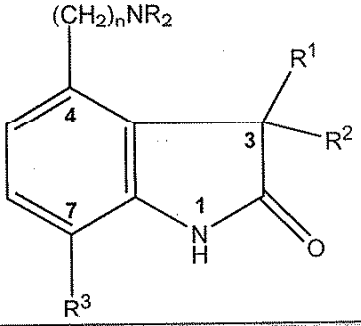
14/ The examples provided in the '860 patent consist largely of the experimental results contained in a report issued by GSK's Department of Preregistrational Affairs—SK&F Report No. PW005BA (DTX 35). As described above, Bradford researchers performed those experiments in consultation with GSK after Dr. Owen shared with Dr. Costall his hypothesis about ropinirole's anti-Parkinson's potential. *See* paragraphs 33-41, *supra*. The work was prepared under contract for GSK, and Bradford was required to turn over the results of their work to GSK. *See* paragraph 35, *supra*.

patents and concluded that the claim 1 of the '860 patent is, in many respects, "much narrower than the genus claims, than the genuses claimed by the '944 and the '808 patent." Trial Tr. 489:11-12 (Bartlett).[15/]

57. A side-by-side comparison of the genus claims in the '944, '808, and '860 patents, which is reproduced below, was used at trial as PDX 27 to aid Dr. Bartlett's testimony:

15/ Teva challenges Dr. Bartlett's qualifications to testify on this subject, *see* Teva's Proposed Findings ¶ 51, but failed to raise any timely challenge to Dr. Bartlett's testimony in accordance with this Court's pre-trial procedures. Accordingly, Teva's challenge to Dr. Bartlett's qualifications is waived.

COMPARISON OF GENUS CLAIMS IN '944, '808, & '860 PATENTS

'944 Patent	'808 Patent	'860 Patent
		
R	**R**	**NR_2**
Amino	Amino	Amino (when both **R**'s = H)
Lower alkylamino	C_{1-6} lower alkylamino	C_{1-4} alkylamino (when one **R** = H and the other **R** = C_{1-4}-lower alkyl)
di-loweralkylamino	di-(C_{1-6}-loweralkyl)amino	di-(C_{1-4}-loweralkyl)amino (when both **R**'s = C_{1-4}-lower alkyl)
	allylamino	
di-N-allylamino	diallylamino	
N-allyl-N-lower alkylamino	N-(C_{1-6}-lower alkyl)-N-allylamino	
	benzylamino, dibenzylamino	
	phenethylamino, diphenethylamino	
	4-hydroxyphenethyl amino, di-(4-hydroxyphenethyl)amino	
R^1	**(position not designated)**	**R^3**
Hydroxy (OII)		Hydroxy
Methoxy (OCH_3)		
	Hydrogen	Hydrogen
(position not designated)	**R^1**	**(position not designated)**
Hydrogen	Hydrogen	Hydrogen
	C_{1-4}-lower alkyl	
(position not designated)	**R^2,R^3 (independently)**	**R^1,R^2 (independently)**
Hydrogen	Hydrogen	Hydrogen
	C_{1-4}-lower alkyl	C_{1-4}-lower alkyl
N	**n**	**n**
1-3	1-3	1-3

58. As illustrated by this chart, the genus claim in the '808 patent—which is listed on the face of the '860 patent as a reference cited during patent prosecution, *see* PTX 35—is substantially broader in many respects than Claim 1 of the '860 patent.[16/] For example, the scope of the substituents at position R in the '860 genus is much narrower than those at position NR2, which is the corresponding position in the '808 genus. Specifically, the '808 genus includes a number of substituents at this position, such as allylamino, diallylamino, benzylamino, dibenzylamino, phenethylamino, and diphenethylamino, that do not appear at the corresponding position in the '860 patent. *See* Trial Tr. 489:17-490:17 (Bartlett). Moreover, the '808 genus allows for alkyl groups of between C1 and C6, whereas the '860 genus is limited to alkyl groups of between C1 and C4. *See id.*[17/] Teva's pharmacology expert, Dr. Long, admitted that the '860 genus claim was narrower than the'808 genus claim in this respect:

> Q: Dr. Long, the group that claims 1 to 4 is narrower than the group that claims 1 to 6. Correct?
>
> A: Correct.
>
> Q: The one that claims1 to 4 is the '860 patent. Correct?
>
> A: Well, 1 to 4 would also be included in the '808 patent.
>
> Q: But the narrower claimed group is the '860 patent. Correct?
>
> A: Yes.

Trial Tr. 234:18-235:2 (Long).

59. While Teva continues to pursue its inequitable conduct challenge to the '860 genus claim, it has abandoned its similar challenge to the '808 genus claim notwithstanding the

---

16/ Although the vast majority of the compounds in the '860 genus claim appear in the '944 and/or the '808 genus claim, claim 1 of the '860 patent does, in at least one instance, contain a combination of substitutents that individually can be found in either of the two prior patents but was not combined until the '860 patent. *See* Trial Tr. 507:24-508:4 (Bartlett).

17/ "An alkyl group is about the simplest molecular fragment, simplest class of molecular fragments in organic chemistry." Trial Tr. 490:18-21 (Bartlett). It consists of carbons and hydrogens and a single bond. *See* Trial Tr. 491:4-25 (Bartlett).

greater breadth of that claim with respect to the alkyl groups. *See* Stipulation to Partially Dismiss Claims Related to U.S. Patent No. 4,452,808 (Without Prejudice) entered December 14, 2006 [D.I. 162]. Teva's pharmacology expert, Dr. Long, conceded this point at trial:

> Q: Now, the '808 patent, which has the broader group – correct?
>
> A: Yes.
>
> Q: You are not challenging that genus, are you?
>
> A: No.
>
> Q: So you are not challenging the broader one, just the narrower one. Correct?
>
> A: Correct.

Trial Tr. 235:3-10 (Long).

60. Dr. Bartlett offered the opinion that the scope of the genus claim in the '860 patent was "quite reasonable" based on the scope of the genus claims for indolone compounds that were in the art at that time. Trial Tr. 486:23-487:11 (Bartlett). Dr. Long did not offer a contrary opinion.

61. Teva's suggestion that GSK's position concerning the reasonableness of claim 1 of the '860 patent is in tension with GSK's position concerning the non-obviousness of the claim 3 of the patent, *see* Teva Proposed Inequitable Conduct Findings ¶ 93, is inaccurate. The '808 patent discloses a genus of indolone compounds, including ropinirole, that act as *peripheral* D_2 agonists and have utility in treating cardiovascular disorders. *See* '808 Patent, Col. 4, ll. 31-44; Col. 1, ll. 46-48; Col. 4, ll. 26-29 (PTX 13); Trial Tr. 327:6-11 (Long); 556:23-557:16 (Jenner). Prior to Dr. Owen's invention, the only reported information about the indolones claimed in the '808 patent suggested that they did not have central behavioral effects. *See* 1985 Gallagher article at 1535 (PTX 17). Once Dr. Owen observed central nervous system effects of ropinirole and concluded that it could be useful in treating Parkinson's Disease, it was perfectly

reasonable to infer that the other indolone compounds disclosed in the '808 patent and the prior art '944 patent would behave similarly.[18/]

*c) **No Known Inactivity***

62. Teva also challenges the '860 genus claim on the basis that Teva's pharmacology expert, Dr. Long, identified two examples of compounds (among the 750 compounds allegedly encompassed by claim 1) that he "wouldn't *expect* activity with." Trial Tr. 219:8-13 (Long) (emphasis added); *see also* Teva Proposed Inequitable Conduct Findings ¶¶ 45-49, 103. Teva, however, offers no evidence that any of the claimed compounds was known by GSK at the time to be inactive in treating Parkinson's Disease.

(1) Cannon 1978 Article (DTX 160)

63. Relying on the speculative testimony of Dr. Long, Teva suggests that the compounds in claim 1 of the '860 patent in which a dibutyl group is added to the aminoethyl side chain would be ineffective treatments for Parkinson's Disease. *See* Teva Proposed Inequitable Conduct Findings ¶¶ 48-49. In attempting to support this argument, Teva relies on a 1978 publication by Cannon, Long, and others titled *Preparation and Biological Actions of Some Symmetrically N,N-Disubstituted Dopamines*, J. Med. Chem., 21(3), 248-53 (1978) ("1978 Cannon article") (DTX 160). Teva's challenge to the dibutyl substitutions in the '860 genus claim is unsupported by the record.

18/ Moreover, Teva's argument confuses one of ordinary skill in the art with the inventor. If, as Teva argues, there is no invention unless one of ordinary skill in the art could form an expectation as to the compound's activity, *see* Teva Proposed Inequitable Conduct Findings ¶ 93, then all inventions would fail on the grounds of obviousness. The Federal Circuit has noted repeatedly that the knowledge of an inventor may not be substituted for the knowledge of one of ordinary skill in the art. *See Standard Oil Co. v. Am. Cyanamid Co.*, 774 F.2d 448, 454 (Fed. Cir. 1985) (noting that the "actual inventor's skill is irrelevant" to the obviousness inquiry, which requires a determination of the scope and content of the prior art); *Bausch & Lomb, Inc. v. Barnes-Hind/Hydrocurve, Inc.*, 796 F.2d 443, 447-48 (Fed. Cir. 1986) (same).

64. The 1978 Cannon article is inapplicable to the indolone compounds described in claim 1 of the '860 patent. The article addresses dopamine derivatives, which is a different class of compounds from the indolones claimed in the '860 patent. *See* 1978 Cannon article (DTX 160); Trial Tr. 592:1-4 (Jenner); Trial Tr. 173:23-174:4 (Long); *see also* Trial Tr. 496:13-497:1 (Bartlett). As GSK's pharmacology expert, Dr. Jenner, made clear at trial, the 1978 Cannon Article reveals nothing about the activity of indolones because it addresses a different class of compounds altogether:

> Q: Dr. Jenner, as of 1987, what, if anything, could one of ordinary skill have concluded from this article [DTX 160] about the activity of ropinirole hydrochloride?
>
> A: ***I think one of ordinary skill in the art would not have been able to conclude anything because this is a paper dealing with n,n-disubstituted dopamine derivatives. It is not a paper dealing with indolone derivatives. And I think as we've now already established, you cannot make jumps from one class to another when considering these structure activity relationships.***

Trial Tr. 592:22-593:6 (Jenner) (emphasis added).

65. As acknowledged by Dr. Long during trial, and explained in detail in the GSK Proposed Validity Findings ¶¶ 88-90, it was well-established at the time of the filing of the '860 patent that the activity of one series of compounds could not be used to predict the activity of different chemical series. *See* Trial Tr. 587:8-24; 594:16-595:7 (Jenner); 262:24-264:7; 258:22-259:4 (Long); Cannon, J.G., *The Design of Potential Anti-Parkinsonian Drugs: What is the Dopaminergic Pharmacophore in Ergot Alkaloids?,* Proc. Iowa Acad. Sci., 93(4), 169-74 (1986) ("1986 Cannon article") at 173 (DTX 179).

66. As stated in the 1986 Cannon article, upon which Dr. Long relied in support of the opinions he offered at trial concerning validity, correlations between chemical series could be "meaningless" or even "misleading":

> Different chemical series of dopaminergic agonists may be interacting with the same geographic area on the receptor protein molecule but, depending upon the chemical nature of the specific chemical series of agonists, a different conformation of the receptor protein may be involved. ***Thus, within a given chemical series of agonists, there may be a well defined structure-activity and stereochemical correlation. But, these correlations may disappear when a different chemical series of agonists is addressed, and a new combination of structural parameters and stereochemical requirements may apply. If this be true, structural comparisons and correlations between ergoline derivatives, apomorphine derivatives, and other dopaminergic agonist molecular systems may not only be meaningless, but actually may be misleading.***

1986 Cannon article at 173 (DTX 179) (emphasis added).

67. Accordingly, the Cannon 1978 article, which addresses dopamine derivative compounds that are *not claimed* in the '860 patent, does not demonstrate anything about the activity of the indolone compounds encompassed by claim 1 of that patent. Neither Dr. Long nor any other witness suggested that any *indolone* with a dibutyl group added to the aminoethyl side chain had been tested and shown to be inactive for Parkinson's Disease or anything else.

68. Teva's suggestion that the 1978 Cannon article motivated GSK to omit Dr. Costall (one of the joint authors on the publication) as an inventor on the '860 patent, *see* Teva Proposed Inequitable Conduct Findings ¶ 97, makes no sense. First, as noted above, the 1978 Cannon article reveals nothing about the activity of the compounds claimed in the '860 patent. Second, as described in more detail *infra* (*see* ¶¶ 84-85), there is no evidence that Dr. Costall should have been named as an inventor on the patent. Third, Teva has offered no evidence that anyone involved in the prosecution of the '860 patent knew about the article or Dr. Costall's views about it.[19/] Finally, even assuming that GSK patent attorneys involved in the '860 patent prosecution knew about the article and deemed it relevant (for which there is absolutely no

19/ Indeed, Dr. Costall's views of the 1978 Cannon article are unknown to this day, since Teva failed to ask her about it during her deposition.

record evidence), they would not have been able to skirt the duty to disclose it simply by omitting Dr. Costall as an inventor; as individuals involved in the prosecution, they too were bound by a duty of candor.

(2) The DeMarinis Article (DTX 56)

69. The only other aspect of the '860 genus challenged by Teva is a single compound referred to in a 1986 article by DeMarinis, *et al.*, as "Compound 31." *See* DeMarinis, et al., *Syntheses and in Vitro Evaluation of 4-(2-Aminoethyl)-2(3H)-indolones and Related Compounds as Peripheral Prejunctional Dopamine Receptor Agonists*, J. Med. Chem., 929, 939-47 (1986) (the "1986 DeMarinis article") (DTX 56); Teva Proposed Inequitable Conduct Findings ¶¶ 47, 103, 106. There is no record support for Teva's suggestion that GSK knew at the time of patent prosecution that Compound 31 would not be effective in treating Parkinson's Disease.

70. The 1986 DeMarinis article focuses on the peripheral activity of ropinirole and other indolone compounds, noting their potential for treating cardiovascular indications. *See* DeMarinis article at 939-40 (DTX 56). Unlike the 1985 Gallagher article (PTX 17), which contains a statement concerning the absence of central behavioral effects of ropinirole and, hence, was disclosed as prior art in the '860 patent (PTX 35), the 1986 DeMarinis article says nothing about the *central* effects of ropinirole or any other indolone compound. Because it focuses exclusively on peripheral effects of indolone compounds (including Compound 31), the 1986 DeMarinis article and its discussion of Compound 31 were not material to the patentability of such compounds for central (i.e., anti-Parkinson's) uses.

71. Furthermore, contrary to Teva's suggestion, the test results on Compound 31 provided in the 1986 DeMarinis article do not demonstrate the compound's inactivity in treating Parkinson's Disease. First, the test used in the article is the rabbit ear artery test. *See* 1986 DeMarinis article at 941 (DTX 56). Teva's pharmacology expert, Dr. Long, acknowledged that,

by 1987, those of ordinary skill in the art knew that the rabbit ear artery test was used to measure activity in the *peripheral* nervous system. *See* Trial Tr. 327:24-328:2; 328:9-11 (Long). GSK's pharmacology expert, Dr. Jenner, agreed and explained that, as of May 1987, one of ordinary skill in the art would not have used the rabbit ear artery test to test for anti-Parkinson's activity. *See* Trial Tr. 564:10-13 (Jenner). Teva's characterization of the results concerning Compound 31 in the 1986 DeMarinis article therefore contradicts the opinions offered by both parties' experts concerning the rabbit ear artery test.

72. Furthermore, the results reported in the 1986 DeMarinis article do not demonstrate that Compound 31 is inactive in the periphery. Rather than reporting zero activity for Compound 31, the article indicates that the compound has activity at a concentration of >3000nm. *See* 1986 DeMarinis article at 943 (DTX 56). The article reports on at least one compound (Compound 47) that requires a higher concentration (>10000 nm) for activity, thereby indicating that there are other compounds that are less active in the periphery than Compound 31. *See id.*; Trial Tr. 494:23-496:5 (Bartlett).[20/]

73. In short, there is absolutely no record evidence that Compound 31 is inactive, either peripherally or centrally. Therefore, Teva's contention that GSK knew that the compound was inactive in treating Parkinson's Disease and deceptively concealed that information from the United States Patent and Trademark Office ("PTO") is baseless.

20/ This fact is not altered by the existence of a 1987 article by Weinstock, et al. describing this compound as "inactive." *See* Weinstock, et al., *Synthesis and Evaluation of Non-Catechol D-1 and D-2 Dopamine Receptor Agonists: Benzimidazol-2-one, Benzoxazol-2-one, and the Highly Potent Benzathiazol-2-one 7-Ethylamines*, J. Med. Chem., 30, 1166-76 (1987) (the "1987 Weinstock article") (DTX 376). The 1987 Weinstock article merely restates the tests from the DeMarinis Article and does not purport to be describing any new or different investigation of the compound, which was not demonstrated to be inactive in the 1986 DeMarinis article. In any case, the 1987 Weinstock article still focuses on the rabbit ear artery test, which Drs. Long and Jenner both testified was a peripheral, rather than central, test. *See* paragraph 71, *supra*; *see also* DTX 376; Trial Tr. 523:12-17 (Bartlett).

6. "Effective Non-toxic Amount"

74. Each of the three claims in the '860 patent involves a method of treatment "which comprises "administering an effective non-toxic amount" of the claimed compounds for the treatment of Parkinson's Disease. *See* '860 Patent, Col. 6, l. 65-Col. 8, l. 14 (PTX 35). The '860 patent does not claim any specific dosage of ropinirole or any other compound, nor did any of the claims in the patent application that led to the patent. *See id*; *see also* DTX 19 at TEV-RQEXP000554. Dr. Owen's testimony that he did not conceive of any *particular* "effective non-toxic amount" of ropinirole, *see* Teva Proposed Inequitable Conduct Findings ¶¶ 30, 43, is unsurprising and entirely consistent with the scope of the patent, which does not claim any such particular dose.[21/]

75. The '860 patent specification provides as background a range of doses that "may be, for example" the daily dose regimen for an adult Parkinson's Disease patient. '860 Patent, Col. 3, ll. 29-39 (PTX 35). Contrary to Teva's suggestion, *see* Teva Proposed Inequitable Conduct Findings ¶¶ 84-85, there is no record evidence that this dosing information was based on Bradford's work. Indeed, a similar hypothetical example about dosing information for hypertensive patients appears in the prior art '808 patent, which Teva is no longer challenging. *See* '808 Patent, Col. 5, l. 59- Col. 6, l. 5 (PTX 13).

21/ In any event, the aspect of Teva's inequitable conduct claim that focuses on the "effective non-toxic amount" language in the '860 patent appears for the first time in Teva's post-trial briefing and should be deemed waived. While Teva did raise an inequitable conduct claim relating to the dosage information and claims in the '808 patent which it abandoned on the eve of trial, Teva made no such claim with respect to the '860 patent. *See* Corrected First Amended Answer, Defenses, and Counterclaims, filed on July 10, 2006 [D.I. 73], at ¶¶ 38, 47, 55-65; Defendant's [Proposed] Findings of Fact and Conclusions of Law, filed on November 3, 2006 [D.I. 137], at ¶¶ 201-204, 208-223.

76. In any event, Teva has not shown that the hypothetical dosing information in the '860 patent specification was material to the patentability of the claimed invention or that there was any intent to deceive the Patent Office in including it in the '860 patent specification.[22/]

7. No Material Misstatements or Omissions Concerning Inventorship

77. Dr. Owen, the sole named inventor on the '860 patent application, submitted a declaration to the PTO stating as follows:

> I believe I am the original, first and sole inventor . . . of the subject matter which is claimed and for which a patent is sought on the invention entitled Medicament the specification of which is attached hereto.
>
> I hereby state that I have reviewed and understand the contents of the above identified specification, including the claims, as amended by any amendment referred to above.
>
> ***
>
> I hereby declare that all statements made herein of my own knowledge are true and that all statements made on information and belief are believed to be true; and further that these statements were made with the knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under Section 1001 of Title 18 of the United States Code and that such willful false statements may jeopardize the validity of the application or any patent issued thereon.

DTX 19 at TEV-RQEXP000555-556.

78. Dr. Owen testified that, to the best of his knowledge, he was the sole inventor, and that he relied on GSK's patent department to make that determination:

> Q: Is there anyone else that you think invented this invention that's described in this 860 patent?

22/ Moreover, to the extent Teva is seeking to bring a back-door enablement challenge to the "effective non-toxic amount" language in the '860 patent through its inequitable conduct claim, such a claim fails for two reasons. First, Teva originally brought an enablement challenge to the patent but abandoned it before trial. *See* Letter of July 17, 2006 from Amy K. Wigmore to Charanjit Brahma (attached hereto as Exhibit G); Letter of July 26, 2006 from Amy K. Wigmore to Charanjit Brahma (attached hereto as Exhibit H). Second, Teva failed to put forth any expert testimony (nor could it) demonstrating that undue experimentation would be required to identify an "effective non-toxic amount" of ropinirole.

> A: I understand entirely that inventorship is a legal matter. To the best of my knowledge, and I don't have that legal expertise, nobody else is an inventor
>
> Q: What is your understanding of what the term inventorship means?
>
> MS. WIGMORE: I'm going to object, calls for a legal conclusion. Answer if you're able.
>
> A: I'm going to – I'd like to answer with a context. Although I can't remember the details of everything that was done, it was my nature and my style to be transparent. I disclosed information to the patent department, and in my view, quite correctly, the patent department were the determinants of who was the inventor.

Owen Dep. Tr. 80:14-81:9 (Ex. C).

79. Dr. Owen further testified that he believed his declaration concerning inventorship (DTX 79) was accurate when he signed it:

> Q: There's only one inventor named on the patent, correct?
>
> A: Correct.
>
> Q: So am I accurate in understanding that at the time you signed this declaration, you believed that you were the original first and sole inventor of the invention, or of the subject matter that's claimed in the 860 patent?
>
> A: That is what I believed, and the reason I believed it is, as has been said, it's a legal judgment, and that was the advice given to me by the patent department.

Owen Dep. Tr. 82:19-83:8 (Ex. C).

80. Teva's suggestion that Dr. Owen did not review the '860 patent application before signing the declaration, *see* Teva Proposed Inequitable Conduct Findings ¶ 40, is a mischaracterization of Dr. Owen's testimony. When asked during his deposition – which took place more than 19 years after he submitted the declaration at issue – whether he had reviewed the U.S. or U.K. patent applications before the '860 patent issued, Dr. Owen replied that he did not "*absolutely* remember" and did not "remember it as an *absolute fact*." *See* Owen Dep. Tr. 31:14-22 (Ex. C) (emphasis added). Especially in light of the contemporaneous sworn

declaration (made under penalty of fine or imprisonment) indicating that Dr. Owen had reviewed the '860 specification and claims, his lack of recollection concerning the specific circumstances surrounding the submission of the application almost 20 years after the fact does not provide any basis for inferring misconduct.

81. Nor does Dr. Owen's reliance on GSK's patent attorneys to prepare the patent application and determine the scope of the claims and inventorship, *see* Owen Dep. Tr. 25:21-27:6; 80:14-81:9; 82:19-83:8; 131:13-132:12 (Ex. C), evidence improper conduct on his or GSK's part. Relying on attorneys to make legal determinations that form the basis for certifications is perfectly reasonable. Indeed, Teva's corporate representative, Deborah Jaskot, acknowledged that she similarly relied on lawyers when she executed the Paragraph IV certification that prompted this lawsuit and that she personally did not even read the patents that she certified were invalid, unenforceable, or not infringed. *See* Trial Tr. 88:23-90:20 (Jaskot). Similarly, Teva's pharmacology expert, Dr. Long, acknowledged that he was a named inventor on patents containing genus claims and that he had no idea how the specific claims came to be but, instead, relied on others. *See* Trial Tr. 368:19-370:14 (Long).

82. At bottom, Teva has failed to adduce any evidence, much less clear and convincing evidence, that Dr. Owen or GSK made any material misrepresentations or omissions concerning inventorship with an intent to deceive. Dr. Owen stated his belief that he was properly named as the sole inventor on the '860 patent, and not a single witness contradicted this view.

83. Teva's newly-minted contention that Annette Wright, Dr. Owen's lab technician, should have been named as an inventor lacks any support in the record.[23/] As discussed further above, Ms. Wright merely conducted cardiovascular experiments under Dr. Owen's direction and shared with him behavioral observations that he observed himself and ultimately relied on in forming his hypothesis that ropinirole was a potential anti-Parkinson's agent. *See* paragraphs 21-32, *supra*. While there is some evidence that Ms. Wright thought that stereotypy could indicate central nervous system effects, *see* Teva's Proposed Inequitable Conduct Findings ¶ 20, there is not a shred of evidence in the record indicating that Ms. Wright ever suggested using ropinirole for the treatment of Parkinson's Disease. Indeed, her laboratory notebook makes no reference to Parkinson's Disease or central nervous system effects in connection with ropinirole. *See generally* DTX 24. Moreover, not a single witness identified Ms. Wright as someone who should have been named an inventor, and Teva did not even seek to depose her to ask that question. Finally, given that Ms. Wright was a GSK employee just like Dr. Owen at the time of the invention, there is no evidence of any motive on GSK's part to conceal any role she may have played in the invention.

84. Teva's efforts to suggest that Dr. Costall and Bradford were improperly omitted as inventors are equally without support. Dr. Costall's testimony is absolutely consistent with Dr. Owen's – i.e., that Dr. Owen presented her with an already-formed hypothesis about ropinirole's potential use as an anti-Parkinson's agent and asked that she and her colleagues perform experiments to confirm that potential. *See* paragraphs 28-41, *supra*. Thus, there is no evidence that any of Bradford's work, which is incorporated into an *internal* GSK report (SK&F Report No. PW005BA (DTX 35), constitutes prior art under 35 U.S.C. § 102(f). *Supra*

23/ This argument should be deemed waived given Teva's failure to raise it before and during the trial of this matter.

paragraph 38. Finally, there is no evidence of any motive on GSK's part to conceal any role Bradford may have played in the invention given that Bradford assigned its research results and any associated intellectual property rights to GSK. *See* paragraph 35, *supra*.[24/]

85. The testimony of Dr. Carol Harvey further corroborates Dr. Owen's conception of the idea of using ropinirole to treat Parkinson's Disease. *See* paragraph 32, *supra*. Moreover, the testimony of Roger Eden, a former GSK pharmacologist, does not in any way undermine Dr. Owen's status as the sole inventor of the subject matter claimed in the '860 patent. While Teva asserts that "Mr. Eden does not credit Dr. Owen with discovery that ropinirole had anti-Parkinson's effects," *see* Teva Proposed Inequitable Conduct Findings ¶ 29, that is misleading. The testimony that Teva cites, but does not quote, in support of that proposition reads as follows:

> Q: Okay. Who first – to your knowledge, who first thought that ropinirole could be used to treat Parkinson's disease?
>
> A: *I don't know.*

Eden Dep. Tr. 94:14-17 (Ex. D) (emphasis added). Thus, far from contradicting the inventorship designation in the '860 patent, Mr. Eden simply disclaims any knowledge of who the inventor was. Mr. Eden's lack of knowledge is unsurprising in light of his testimony that he "wasn't privy to all the conversations and telephone calls," including the initial contact with Bradford. Eden Dep. Tr. 60:6-9 (Ex. D); *see also id.* 58:2-12 (Ex. D).

86. Teva has also failed to identify any improper conduct on GSK's or Dr. Owen's part concerning the '860 genus claim (claim 1). Dr. Giddings – the only currently-employed GSK patent attorney who had any role in the prosecution of the application that became the '860 patent – expressly denied that he was an inventor of claim 1. *See* paragraph 54, *supra*. Teva has

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24/ As discussed in paragraph 68 above, the 1978 Cannon Article also does not support any motive on GSK's part to conceal any role played by Dr. Costall or Bradford.

also provided no evidence that the Honorable Alan Lourie or Stuart Suter (the two GSK patent attorneys named on the '860 patent, who were never deposed by Teva) or Vincent Fabiano (a former GSK patent attorney who was identified in GSK's interrogatory responses, *see* Teva Proposed Inequitable Conduct Findings ¶ 59 and DTX 140, but never deposed by Teva) played a role in the invention and deceptively withheld it from the PTO. Nor is there evidence of any motive for concealing the role of any GSK patent attorney in the invention since any such attorney, like Dr. Owen, would have been an employee of the company at the time.

87. Finally, the mere fact that there are unanswered questions about details concerning the prosecution of the '860 patent, *see* Teva Proposed Inequitable Conduct Findings ¶ 5, is unsurprising given the amount of time that has passed and the turnover in GSK's patent department since that time.[25/] At any rate, Teva's list of unanswered questions does not provide evidence of any knowing misstatement or omission with intent to deceive.

C. Allegations Concerning Statements in the '860 Patent Regarding Dopamine Receptors and Prior Art Compounds

88. Teva's allegations that GSK made false statements in the '860 patent concerning the significance of the distinction between pre-synaptic and post-synaptic activity and the characteristics of prior art Parkinson's Disease treatments, *see* Teva Proposed Inequitable Conduct Findings ¶¶ 55-58, also lack factual support. As discussed in more detail below, Teva has failed to carry its burden of demonstrating by clear and convincing evidence that GSK made

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25/ While Teva now takes issue with GSK's privilege instructions, *see* Teva Proposed Inequitable Conduct Findings ¶ 12, Teva never moved to compel in response to any claim of privilege. Furthermore, the testimony of Dr. Owen and Dr. Giddings makes clear that those witnesses have no recollection of the specific circumstances surrounding the '860 patent application, notwithstanding any claim of privilege GSK may have asserted.

any materially false or misleading statement about these issues with an intent to deceive the PTO.

1. Pre-Synaptic v. Post-Synaptic Distinction

89. As described in detail in paragraphs 51-55 of the GSK Proposed Validity Findings, there are different subtypes of dopamine receptors, including D_1 receptors and D_2 receptors. *See* Trial Tr. 540:22-25 (Jenner); 293:10-15 (Long). Both D_1 and D_2 receptors can be found in both the peripheral and central nervous systems. *See* Trial Tr. 541:9-15 (Jenner). Dopamine receptors can be located on the pre-synaptic side of a nerve cell (before the synapse) and on the post-synaptic side of a nerve (after the synapse). *See* Trial Tr. 540:9-21 (Jenner); 165:9-15 (Long).

90. The claims in the '860 patent (including claim 3, which is the only claim asserted by GSK in this action) make no reference to the specific receptors or receptor sites with which the claimed compounds interact. Instead, the patent simply claims the use of those compounds for the treatment of Parkinson's Disease. *See* '860 Patent, Col. 6, l. 66-Col. 8, l. 14 (PTX 35).

91. The specification of the '860 patent contains a background discussion on the treatment of Parkinson's Disease that contains the following statement:

> It has now been found that certain indolone derivatives known in the art as pre-synaptic D_2-agonists having utility as cardiovascular agents (see EP No. 113964-B), also are post-synaptic D_2-agonists in the brain and hence are expected to have utility in the treatment of Parkinsonism.

'860 Patent, Col. 1, ll. 48-53 (PTX 35).

92. Teva does not challenge the accuracy of that statement, nor can it. As Teva's pharmacology expert, Dr. Long, acknowledged, the statement refers specifically to the indolone compounds that are claimed in the patent, including ropinirole. *See* Trial Tr. 366:24-367:14. As explained in paragraph 16 above and described in more detail in paragraphs 77-81 of the GSK

Proposed Validity Findings, the prior art '808 patent describes ropinirole and other indolone derivatives as having activity at *pre-synaptic peripheral D_2 receptors* and utility as dopamine agonists in treating disorders of the cardiovascular system. Prior to the '860 patent application, there was no suggestion that ropinirole or any other indolone compound had post-synaptic activity at central D_2 receptors: indeed, the '860 patent noted that the prior art 1985 Gallagher article indicated that "such compounds have previously been reported as not being capable of producing the central behavioural effects often seen with dopamine agonists." '860 Patent, Col. 1, ll. 54-58 (PTX 35), citing 1985 Gallagher article (PTX 17).

93. Moreover, Dr. Owen's hypothesis that ropinriole could be used to treat Parkinson's Disease, *see* paragraphs 26-27, 31-33 above, necessarily presumed its interaction with *post-synaptic central D_2 receptors*. Dr. Owen and the two pharmacology experts who testified at trial all agreed that a compound must interact with post-synaptic central D_2 receptors in order to treat Parkinson's Disease. *See* Trial Tr. 320:11-15 (Long); 546:14-19 (Jenner); Owen Dep. Tr. 192:13-193:3 (Ex. C).

94. As described above, in order to confirm Dr. Owen's hypothesis, Bradford scientists performed experiments on ropinirole in consultation with GSK at Dr. Owen's request. *See* paragraphs 28-41, *supra*. The results of those experiments, which are included in a GSK report (SK&F Report No. PW005BA) and the '860 patent, confirm ropinirole's interaction with post-synaptic D_2 receptors. *See* SK&F Report No. PW005BA at GSK-REQ001030 (DTX 35); '860 Patent, Col. 4, ll. 1-24 (PTX 35).

95. Contrary to Teva's assertion, *see* Teva Proposed Inequitable Conduct Findings ¶¶ 57-58, 116, 119, Dr. Owen does not dispute the truthfulness of the statement made in the '860

patent regarding ropinirole's pre-synaptic and post-synaptic activity. In his deposition, he instead acknowledged the appropriateness of the statement made:

> Q: Let me go up a little bit, I'm looking at your patent again, exhibit 17, we were previously looking at column 1, the paragraph starting at line 54, I'm going to ask you to go to the paragraph above that, which says: "It has now been found that certain indolone derivatives, known in the art as presynaptic D2 agonists, having utility as cardiovascular agents [and then it cites a European patent] also are postsynaptic D2 agonists in the brain and hence are expected to have utility in the treatment of Parkinson's."
>
> Do you see that?
>
> A: Yes, I do.
>
> Q: Is that an accurate assessment of what you hypothesized when you first spoke to -- before you first spoke to Dr. Costall?
>
> A: ***That's an accurate reflection of what would be appropriate if the hypothesis was correct,*** but if you're asking -- I need to be sure what you're asking me, because that's not the language I would have used with Professor Costall.

Owen Dep. Tr. 189:17-190:16 (Ex. C) (emphasis added). Dr. Owen's testimony makes clear that he was relying on the experiments performed by the Bradford scientists to confirm his hypothesis, which is exactly what they did. *See* paragraph 36, *supra*.

96. Teva also mischaracterizes Dr. Owen's testimony concerning the relevance of the distinction between pre-synaptic and post-synaptic receptors. *See* Teva Proposed Inequitable Conduct Findings ¶¶ 57-58, 119, 122. While Teva quotes selectively from an answer given by Dr. Owen, the entire answer, when viewed in context, makes quite clear that Dr. Owen was *not* saying that pre-synaptic versus post-synaptic activity is irrelevant for purposes of treating Parkinson's disease. Instead, he expressly acknowledged that a compound must be an agonist at post-synaptic receptors to treat Parkinson's Disease:

> Q: So you knew that it was -- that ropinirole was a D2 agonist, to you it didn't matter whether it had an effect on presynaptic D2 receptors or postsynaptic D2 receptors, in order to be effective for Parkinson's disease?

> MS. WIGMORE: Objection.
>
> A: ***In order to be effective in Parkinson's disease, the evidence that we have now is that it is an agonist at postsynaptic receptors,*** but this --whether it's pre or postsynaptic, in my view, is an absolute irrelevance. A D2 agonist is a D2 agonist for D2 receptors regardless of their location.

Owen Dep. Tr. 192:13-193:3 (Ex. C) (emphasis added). An examination of this complete excerpt in combination with the testimony surrounding it makes clear that what Dr. Owen was conveying in the latter part of the sentence is that a compound's activity at pre-synaptic versus post-synaptic receptors has no bearing on whether it will be a D_2 agonist; instead, in Dr. Owen's own words:

> Q: So a compound will be a D2 receptor agonist regardless of whether the receptor is presynaptic or postsynaptic?
>
> MS. WIGMORE: Objection. Go ahead.
>
> A: If it's an agonist for D2 receptors, it will be an agonist for D2 receptors, and whether they are presynaptic or postsynaptic is a piece of anatomy, not a piece of pharmacology.

Owen Tr. Dep. 191:7-14; *see also id.* at 191:19-21 ("If a compound is a D2 agonist, I would expect it to be a D2 agonist at D2 receptors regardless of their anatomical location") (Ex. C).

97. In sum, there is no record evidence supporting Teva's assertion that the distinction between pre-synaptic and post-synaptic activity made in the background section of the '860 patent specification (Col. 1, ll. 48-53 (PTX 35)) is false or inconsistent with Dr. Owen's hypothesis. There is also no evidence that these background statements, which simply reiterate a distinction that was well understood by those of ordinary skill in the art at the time and is not part of the patent claims, *see* paragraphs 90-93 above, was material to the patentability of the claimed subject matter. Finally, Teva has offered absolutely no evidence of any intent by GSK or Dr. Owen to deceive the PTO with respect to this background statement. At best, Teva has shown that Dr. Owen has an incomplete recollection of the details underlying statements in the '860

patent, which is understandable given the nearly twenty years that have elapsed since the application was filed. *See, e.g.*, Owen Dep. Tr. 98:18-99:4 ("I don't know the answer to that. I don't remember what I knew at that level of detail at that time."); 122:12-22 ("I don't recall what happened in detail at that time, therefore, I'm unable to answer that question. As of today, by implication, it's the best part of 20 years since I did active pharmacology; anything what I knew, I only know less now.") (Ex. C).

2. Statements Concerning Bromocriptine

98. The background section of the '860 patent also contains the following statement concerning prior art treatments for Parkinson's Disease, including bromocriptine:

> An alternative form of therapy is to administer post-synaptic dopamine agonists, for example ergot alkaloids such as bromocriptine—however, this approach is also associated with side-effects. For example, patients receiving bromocriptine often experience dyskinesia[,] psychiatric problems, and in a small number of cases experience vasopastic phenomena and angina. In addition bromocriptine also causes psychiatric side-effects such as hallucinations.

'860 Patent, Col. 1, ll. 36-44 (PTX 35).

99. Teva is incorrect in asserting that this statement is false or misleading. *See* Teva Proposed Inequitable Conduct Findings ¶¶ 55-56, 116-118. In fact, during cross-examination at trial, Teva's pharmacology expert, Dr. Long, conceded that this statement was true:

> Q: So the statement in the patent that bromocriptine is post-synaptic and leads to what could be severe side effects was true; correct?
>
> A: Correct.

Trial Tr. 362:15-18 (Long). *See also* Trial Tr. 360:14-361:14 (Long) (agreeing "100 percent" with the statement in the '860 patent that bromocriptine acts post-synaptically).

100. Teva's suggestion that this indisputably true statement is at odds with the 1986 DeMarinis article (DTX 56), *see* Teva Proposed Inequitable Conduct Findings ¶¶ 55-56, 117-

118, is baseless. As explained in paragraph 70 above, the 1986 DeMarinis article focuses solely on *peripheral* dopamine agonist activity, whereas the '860 patent and the statement at issue deal with *central* dopamine agonist activity. Indeed, the title of the 1986 DeMarinis article refers to "*Peripheral* Prejunctional[26/] Dopamine Receptor Agonists." *See* 1986 DeMarinis Article at 939 (DTX 56) (emphasis added). The article says nothing about the effects of ropinirole or any other compound at *central* D_2 receptors, thus making it immaterial to the subject matter claimed in the '860 patent. *See* 1986 DeMarinis Article (DTX 56).

101. The specific statement in the 1986 DeMarinis article that Teva claims is inconsistent with the statement about bromocriptine in the '860 patent reads as follows:

> A number of different chemical structures have demonstrated ***preferential agonist activity at peripheral prejunctional D_2 vis-a-vis postjunctional D_1 receptors. These include for example*** alkylated derivatives of dopamine such as di-n-propyldopamine and n-propyl-n-butyldopamine; cyclized dopamine derivatives of the 2-aminotetralin series and apomorphine; ***ergot alkaloids such as bromocryptine*** [sic] and its simplified derivatives like LY 141865.

1986 DeMarinis article at 940 (DTX 56) (emphasis added). This statement, on its face, compares the activity of bromocriptine at *peripheral* D_2 receptors to its activity at *post-junctional* D_1 receptors. The article says absolutely nothing about bromocriptine's activity, or lack thereof, at post-synaptic D_2 receptors (centrally or peripherally).

102. Teva's expert, Dr. Long, conceded as much:

> Q: Now, the article is talking only about post-junctional D1 receptors; correct?
>
> A: Well, that's what it says.
>
> Q: Right. And when you testified to His Honor about this yesterday, we didn't look at the first portion of the article that tells

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26/ The term "pre-junctional" can be used synonymously with the term "pre-synaptic."

> us what they're [comparing]. They're comparing the peripheral pre-junctional D2 with the post-junctional D1; correct?
>
> A: That's what they say.
>
> ***Q: Right. And there is nothing in the article that says bromocriptine is not post-synaptic; correct?***
>
> ***A: Right.***

Trial Tr. 365:11-22 (Long) (emphasis added).

103. GSK's correspondence with the European Patent Office ("EPO") cited by Teva, *see* Teva Proposed Inequitable Conduct Findings ¶ 56, does not alter the accuracy of the statement in the '860 patent concerning bromocriptine's post-synaptic activity. The correspondence, which was drafted by Dr. Peter Giddings on November 25, 1991, identifies an "inadvertent error" in the description of bromocriptine in the European patent application relating to the '860 patent, and states that "it is correct to state that bromocriptine is a prejunctional D_2 receptor agonist." *See* DTX 133 at GSK-REQ018298. The '860 patent, however, does not say anything about bromocriptine's prejunctional (or presynaptic activity); it simply states that bromocriptine is a *post-synaptic* receptor which, as explained in paragraphs 98-102 above, is indisputably true.

104. Dr. Giddings acknowledged during his deposition that, notwithstanding his statement in the letter to the EPO, he did not see any contradiction between the 1986 DeMarinis article and the portion of the '860 patent specification relating to bromocriptine:

> Q: If you could look at column 1 [of the '860 patent], the paragraph starting at line 36.
>
> A: Yes.
>
> Q: Do you see where it says, "An alternative form of therapy is to administer postsynaptic dopamine agonists, for example ergot alkaloids such as bromocriptine?" Do you see that?
>
> A: Yes.
>
> Q: The characterization there of ergot alkaloids, like bromocriptine as postsynaptic dopamine agonists, does that

> contradict the portion of the DeMarinis article that we were just looking at?
>
> MS. WIGMORE: Objection.
>
> THE WITNESS: Well, the sentence in the DeMarinis article, it says it's prejunctional D2 receptor agonists.
>
> BY MR. BRAHMA: Prejunctional, correct.
>
> THE WITNESS: Prejunctional, yes. ***But I don't see that it says it's not a postjunctional receptor agonist.*** Well, that would be my observation as I sit here.

Giddings Dep. Tr. 143:12-144:10 (Ex. F).

105. At most, therefore, the 1986 DeMarinis article reveals a characteristic about bromocriptine—that it has *pre-synaptic* dopamine receptor activity in the *periphery*—that was not disclosed explicitly in the background section '860 patent. The absence of any explicit statement of this information in the '860 patent specification does not amount to a material omission.

106. First, it was well-known by those of ordinary skill in the art as of 1987 that bromocriptine interacted with a number of different receptors, including pre-synaptic and post-synaptic dopamine receptors in the peripheral and central nervous systems. *See* Trial Tr. 568:6-11 (Jenner); 361:15-362:14 (Long). Indeed, bromocriptine's interaction with receptors other than post-synaptic central dopamine receptors is implied by the discussion of the side-effects of bromocriptine in the very same paragraph of the '860 patent with which Teva takes issue. The '860 patent notes that bromocriptine has numerous side effects, including some that occur in the periphery such as angina. *See* '860 Patent, Col. 1; ll. 39-42 (PTX 35). Dr. Long acknowledged at trial that one of ordinary skill in the art would have been well aware that the side effects of bromocriptine would have been caused by bromocriptine's interaction with different receptor binding sites:

Q: The rest the paragraph describes side effects associated with bromocriptine?

A: Yes.

Q: Now, you agree there are side effects associated with bromocriptine?

A: Yes.

Q: And the reason there are side effects is bromocriptine is not a particularly clean compound, is it?

A: That's correct.

Q: And it can bind with a lot of different receptor sites; correct?

A: Correct.

Q: And because it can bind with a lot of different receptor sites, you can get these side effects; correct?

A: Yes, I presume so. Yes.

Q: And one of ordinary skill in the art would have known in 1987 that bromocriptine was not a clean compound; correct?

A: Yes. May I comment a moment here?

Q: Well, Dr. Long --

A: They would have known it was a nonclean compound.

Q: Okay. And would have known, the fact that it was not a clean compound would lead to the possibility of side effects; correct?

A: Correct.

Trial Tr. 361:15-362:14 (Long).

107. Second, bromocriptine's status as both a pre-synaptic and post-synaptic dopamine agonist had no bearing on the patentability of the invention claimed in the '860 patent. As discussed in detail in paragraphs 70 through 73 of GSK Proposed Validity Findings, the fact that a given compound had activity at both pre-synaptic peripheral receptors and post-synaptic central receptors would not have enabled one of ordinary skill in the art to make any prediction about whether ropinirole would behave similarly. It was believed at the time that there were also compounds that interacted with pre-synaptic peripheral D_2 receptors *but not* post-synaptic central

D_2 receptors. *See* Trial Tr. 560:18-22 (Jenner); 302:6-15 (Long). Moreover, Teva's expert, Dr. Long, acknowledged that one would need to test any given compound to determine whether it had both pre-synaptic and post-synaptic activity. *See* Trial Tr. 355:21-356:3 (Long).

108. In particular, as Dr. Jenner made clear in his testimony, because bromocriptine is part of a different chemical series than the indolone compounds claimed in the '860 patent, one of ordinary skill in the art could not form any view about ropinirole's activity at dopamine receptors based on that of bromocriptine:

> Q: What would one of ordinary skill in the art have known in May of 1987 about bromocriptine's interaction with dopamine receptors?
>
> A: Bromocriptine was known to interact with both pre-synaptic and post-synaptic dopamine receptors in the peripheral and central nervous systems.
>
> * * * *
>
> Q: ***What if anything would one of ordinary skill in the art have concluded about ropinirole's activity at dopamine receptors based on this knowledge about bromocriptine as of May of 1987?***
>
> A: ***Well, I don't think one would have concluded anything, quite frankly, because ropinirole is an indolone compound, bromocriptine is a complex ergot derivative. And I think, as we have heard, and certainly as Drs. Cannon and Long have [taught], you cannot transpose information between one chemical class of dopamine agonists and another.***

Trial Tr. 568:6-11; 568:19-569:3 (Jenner) (emphasis added). *See also* paragraph 66, *supra*; 1986 Cannon article at 173 (DTX 179).

109. Furthermore, bromocriptine's interaction with both peripheral pre-synaptic receptors and central post-synaptic receptors would not have resulted in an expectation that the indolone compounds could be used as anti-Parkinson's drugs in light of the prior art 1985 Gallagher article (PTX 17), which is discussed *supra* at paragraph 20. Dr. Giddings made this point clearly in the correspondence with the EPO upon which Teva relies:

> Applicants do not, however, accept that it is the case that the use of the present compounds as anti-Parkinson's drugs is to be expected in view of the disclosure in [the 1985 Gallagher article] . . . which teaches ***against*** the use of such compounds in diseases of the central nervous system.

See DTX 133 at GSK-REQ-018298 (emphasis added). The EPO presumably accepted this argument when it issued the patent GSK was seeking, notwithstanding its awareness of bromocriptine's activity at both pre-synaptic and post-synaptic dopamine receptors. Giddings Dep. Tr. 103:8-19 (Ex. F)

110. Finally, Teva has not offered any evidence suggesting that GSK's attorneys or Dr. Owen made knowing misstatements or omissions concerning bromocriptine with the intent to deceive the PTO. To the contrary, the correspondence on which Teva relies identifying this alleged error was written in November 1991 – long after the '860 patent application was filed and the patent was issued. *See* DTX 133 at GSK-RQ018298. Moreover, the document states on its face that any error with respect to the statement at issue was "inadvertent." *See id.*

CONCLUSIONS OF LAW

I. LEGAL STANDARDS

A. Inequitable Conduct

111. Applicants for patents have a duty to prosecute patents in the United States Patent and Trademark Office ("PTO") with candor and good faith, including a duty to disclose information known to the applicants to be material to patentability. A breach of this duty may constitute inequitable conduct, which can arise from an affirmative misrepresentation of a material fact, failure to disclose material information, or submission of false material information, coupled with an intent to deceive or mislead the PTO.

112. "A party asserting that a patent is unenforceable due to inequitable conduct must prove materiality and intent by clear and convincing evidence. Once threshold findings of materiality and intent are established, the trial court must weigh them in order to determine whether the equities warrant a conclusion that inequitable conduct occurred. This requires a careful balancing: when the misrepresentation or withheld information is highly material, a lesser quantum of proof is needed to establish the requisite intent. In contrast, the less material the information, the greater the proof must be." *Purdue Pharma L.P. v. Endo Pharm., Inc.*, 438 F.3d 1123, 1128-29 (Fed. Cir. 2006) (citations omitted).

113. Information is material "when it is not cumulative to information already of record or being made of record in the application, and

(1) it establishes, by itself or in combination with other information, a prima facie case of unpatentability of a claim; or

(2) it refutes, or is inconsistent with, a position the applicant takes in:

(i) opposing an argument of unpatentability relied on by the Office, or

(ii) asserting an argument of patentability.

37 C.F.R. § 1.56(b); *Purdue Pharma*, 438 F.3d at 1129. Information is deemed material if there is a substantial likelihood that "a reasonable examiner would have considered such prior art important in deciding whether to allow the patent application.'" *Dayco Prods., Inc. v. Total Containment, Inc.*, 329 F.3d 1358, 1363 (Fed. Cir. 2003) (quoting *Driscoll v. Cebalo*, 731 F.2d 878, 884 (Fed. Cir. 1984)).

114. If "threshold levels of materiality and intent are satisfied, the ultimate determination of inequitable conduct is within the discretion of the trial court, which must make the equitable judgment concerning whether the applicant's conduct is so culpable that the patent should not be enforced." *Life Techs., Inc. v. Clontech Labs., Inc.*, 224 F.3d 1320, 1324 (Fed. Cir. 2000); *see also Specialty Composites v. Cabot Corp.*, 845 F.2d 981, 993 (Fed. Cir. 1988) (finding no inequitable conduct even if threshold levels of intent and materiality are demonstrated).

B. Inventorship

115. Only the true inventor may apply for and obtain a patent. 35 U.S.C. §101, "Conception is the touchstone to determining inventorship." *Fina Oil & Chem. Co. v. Ewen*, 123 F.3d 1466, 1473 (Fed. Cir. 1997) (citing *Sewall v. Walters*, 21 F.3d 411, 415 (Fed. Cir. 1994)). "Conception is the 'formation in the mind of the inventor, of a definite and permanent idea of the complete and operative invention, as it is hereafter to be applied in practice.'" *Hybritech, Inc. v. Monoclonal Antibodies, Inc.*, 802 F.2d 1367, 1376 (Fed. Cir. 1986) (quoting 1 Robinson on Patents 532 (1890)). "An idea is sufficiently 'definite and permanent' when 'only ordinary skill would be necessary to reduce the invention to practice, without extensive research or experimentation.'" *Ethicon, Inc. v. U.S. Surgical Corp.*, 135 F.3d 1456, 1460 (Fed. Cir. 1998) (quoting *Burroughs Wellcome Co. v. Barr Labs., Inc.*, 40 F.3d 1223, 1228 (Fed. Cir. 1994)).

116. "Reduction to practice," involves a showing that the invention "actually worked for its intended purpose." *DSL Dynamic Sciences Ltd. v. Union Switch & Signal, Inc.*, 928 F.2d 1122, 1125 (Fed. Cir. 1991).

117. "An inventor need not know that his invention will work for conception to be complete. He need only show that he had the idea; the discovery that an invention actually works is part of its reduction to practice." *Burroughs Wellcome Co.*, 40 F.3d at 1228 (citing *Applegate v. Scherer*, 332 F.2d 571, 573 (C.C.P.A. 1964); *Oka v. Youssefyeh*, 849 F.2d 581, 584 (Fed. Cir. 1988)).

118. Where an inventor has already conceived the idea to use a particular drug to treat a particular condition, inventorship is not negated by use of a third party to confirm activity. Rather, "what matters for conception is whether the inventors had a definite and permanent idea of the operative inventions." *Burroughs Wellcome Co.*, 40 F.3d at 1230. When third party testing "confirm[s] the operability of the inventions" it demonstrates that the original inventor "had a definite and permanent idea of the invention[]." *Id.* Such confirmatory testing is "part of the reduction to practice and inure[s] to the benefit" of the referring inventor. *Id.*

119. The named inventors on an issued patent are presumed to be the true and only inventors. *Hess v. Adv. Cardiovascular Sys., Inc.*, 106 F.3d 976, 980 (Fed. Cir. 1997), *cert. denied*, 520 U.S. 1277 (1997). "Any party wishing to challenge the . . . patent's current inventorship must ultimately come forward with clear and convincing evidence of facts that support its contentions." *Fina Oil & Chem. Co*, 123 F.3d at 1472 (citing *Hess*, 106 F.3d at 979-80).

120. Under 35 U.S.C. §116, joint inventors may apply for a patent. The concept of joint invention has been described as "one of the muddiest concepts in the muddy metaphysics of

the patent law." *Mueller Brass Co. v. Reading Indus.*, 352 F. Supp. 1357, 1372 (E.D. Pa. 1972), *aff'd* 487 F.2d 1395 (3d Cir. 1973). In general, however, "a joint inventor must contribute in some significant manner to the conception of the invention. As such, 'each inventor must contribute to the joint arrival at a definite and permanent idea of the invention as it will be used in practice.'" *Fina Oil & Chem. Co.*, 123 F.3d at 1473 (citations omitted) (quoting *Burroughs Wellcome*, 40 F.3d at 1229).

121. A person does *not* become a co-inventor by: (1) "merely assisting the actual inventor after conception of the claimed invention;" (2) "simply provid[ing] the inventor with well-known principles or explain[ing] the state of the art without ever having a "firm and definite idea' of the claimed [invention] as a whole"; or (3) "simply reduc[ing] the inventor's idea to practice" *Ethicon*, 135 F.3d, at 1460 (citations omitted).

122. "Incorrect inventorship is a technical defect in a patent that may be easily curable." *Canon Computer Sys., Inc. v. Nu-Kote Int'l, Inc.*, 134 F.3d 1085, 1089 (Fed. Cir. 1998). Under 35 U.S.C. § 116, inventorship errors may be corrected by the Patent Office in a pending application. Under 35 U.S.C. § 256, inventorship errors may also be corrected by the Court so long as the error occurred without "deceptive intention." "The court before which such matter is called into question may order correction of the patent on notice and hearing of all parties concerned and the Commissioner shall issue a certificate accordingly." *Id.*

123. Inventorship errors can also be raised as a defense under 35 U.S.C. §102(f), which provides "[a] person shall be entitled to a patent unless he did not himself invent the subject matter sought to be patented." 35 U.S.C. § 102(f). However, "[w]hen a party asserts invalidity under § 102(f) due to nonjoinder, a district court should first determine whether there exists clear and convincing proof that the alleged unnamed inventor was in fact a co-inventor. Upon such a

finding of incorrect inventorship, a patentee may invoke Section 256 to save the patent from invalidity. Accordingly, the patentee must then be given an opportunity to correct inventorship pursuant to that section. Nonjoinder may be corrected "on notice and hearing of all parties concerned" and upon a showing that the error occurred without any deceptive intent on the part of the unnamed inventor." *Pannu v. Iolab Corp.*, 155 F.3d 1344, 1349 (Fed. Cir. 1998) (citing 35 U.S.C. § 256).

C. Generic Claims

124. Patent claims need not be, and rarely are, limited to the specific embodiments disclosed in the patent specification. *See, e.g.*, *In re Fisher*, 427 F.2d 833, 839 (C.C.P.A. 1970) (stating that as long as the specification discloses at least one method for making and using the claimed invention that bears a reasonable correlation to the entire scope of the claim, then the enablement requirement of 35 U.S.C. § 112 is satisfied); *Utter v. Hiraga*, 845 F.2d 993, 998 (Fed. Cir. 1988) ("A specification may, within the meaning of 35 U.S.C. §112 ¶ 1, contain a written description of a broadly claimed invention without describing all species that claim encompasses.").

125. The number and variety of examples are irrelevant if the disclosure is "enabling" and sets forth the "best mode contemplated." *In re Borkowski*, 422 F.2d 904, (C.C.P.A. 1970). A disclosure is enabling even if considerable amount of experimentation is involved, if it is merely routine. *Ex parte Forman*, 230 USPQ 546 (B.P.A.I. 1986). Furthermore, the presence of inoperative or inactive embodiments within the scope of a genus claim does not necessarily render the claim nonenabled. *Atlas Powder Co. v. E.I. du Pont De Nemours & Co.*, 750 F.2d 1569, 1577 (Fed. Cir. 1984). In fact, "[i]t is not a function of the claims to specifically exclude . . . possible inoperative substances . . . ." *Id.* (quoting *In re Dinh-Nguyen,* 492 F.2d 856, 858-59 (C.C.P.A. 1974)). Potentially inoperative species are only non-enabling "if the number of

inoperative combinations becomes significant, and in effect forces one of ordinary skill in the art to experiment unduly in order to practice the claimed invention." *Id.* (citing *In re Cook,* 439 F.2d 730, 735 (1971)).

126. Among the duties of the patent practitioners before the PTO is the obligation to obtain the broadest claims possible that meet the requirements of Title 35, consistent with the disclosure of the patent application, the scope of the prior art of which he or she is aware, and the duty of candor. *See, e.g.*, Robert C. Faber, *Landis on Mechanics of Patent Claim Drafting* § 10:1.1 (5th ed. 2005); Jeffrey G. Sheldon, *How to Write a Patent Application*, Practicing Law Institute § 6.5.3 (2006); Irving Kayton, *Kayton on Patents* 3-1 (2d ed. 1983), 3-1 (quoted *supra* at paragraph 53).

127. With respect to chemical patents in particular, a patent limited to the precise compounds reduced to practice by an inventor would frequently be of little or no value because of the ability to obtain the same functionality of the compound by making minor substituent variations or manipulations to the molecule. Thus, it is normal practice for a patent attorney to draft a patent application to include generic formulas that would include any related substituents that could reasonably be expected to exhibit similar activity and to seek claims to such generic formulas. This does not make the inventor of the chemical compound any less the inventor of the generic formula. Rather, invention of the specific compound entitles the inventor to a genus of compounds of reasonable scope. *See, e.g., Enzo Biochem, Inc. v. Gen-Probe Inc.,* 323 F.3d 956, 974 (Fed. Cir. 2002) (Lourie, J., concurring in decision to deny rehearing en banc) ("Although one may envision a general concept, what one usually does first in making or isolating a chemical or chemical-related invention is to obtain a specific material or materials. One then broadens the concept to extend it as far as one envisions that other materials will have

the same utility and can be similarly made. That broadened concept becomes the genus in a patent application that is both the broadest statement constituting a written description and usually claim 1.").

II. ULTIMATE CONCLUSIONS OF LAW AND UNDERLYING FACT

128. Teva has failed to carry its burden of showing by clear and convincing evidence that the '860 patent was procured by inequitable conduct. Accordingly, there is no basis for Teva's request that the '860 patent be deemed unenforceable.

A. Inventorship

129. Teva has failed to carry its burden of demonstrating by clear and convincing evidence that the '860 patent was obtained through inequitable conduct relating to inventorship.

130. David Owen is the named inventor on the '860 patent. Accordingly, he is presumed to be the true and only inventor, and Teva can only rebut this presumption with clear and convincing evidence. *Hess*, 106 F.3d at 979-80; *Fina Oil & Chem. Co.*, 123 F.3d at 1472. For the reasons described in detail above and summarized in paragraphs 77-87, Teva has not offered clear and convincing evidence to overcome the presumption that Dr. Owen was correctly named as the sole inventor, that any facts material to the issue of inventorship were misstated or not disclosed to the PTO, or that anyone from GSK involved in the prosecution of the '860 patent acted with an intent to deceive the PTO.

131. Dr. Owen testified that he relied on the expertise of the GSK patent department to draft the '860 patent and to make an inventorship determination and that he believed his inventorship declaration was true when he signed it. *See* paragraphs 77-79, 81, *supra*. Teva has provided no evidence to the contrary. Furthermore, the testimony of Teva's own witnesses, including Ms. Jaskot and Dr. Long, makes clear that reliance on attorneys in the preparation of certifications and patent documents is common practice and entirely reasonable. *See* paragraph 81, *supra*.

132. Teva's allegation that Dr. Costall and the researchers at Bradford were either inventors or co-inventors of the invention claimed in the '860 patent is not supported by the

record. Dr. Costall's testimony is perfectly consistent with that of Dr. Owen and Dr. Harvey in establishing the Dr. Owen came up with the idea of using ropinirole to treat Parkinson's Disease before he approached Bradford. *See* paragraph 33, *supra*. Accordingly, the undisputed evidence refutes Teva's contention that Dr. Owen derived the invention from Bradford within the meaning of 35 U.S.C. §102(f). Moreover, because the invention was already conceived by Dr. Owen before he ever made contact with Bradford, Dr. Costall and her colleagues at Bradford cannot be joint inventors under 35 U.S.C. §116. *See Fina Oil & Chem. Co.,* 123 F.3d at 1473 ("[A] joint inventor must contribute in some significant manner to the conception of the invention. As such, 'each inventor must contribute to the joint arrival at a definite and permanent idea of the invention as it will be used in practice.'") (citations omitted) (quoting *Burroughs Wellcome*, 40 F.3d at 1229). Rather, the Bradford researchers were "merely assisting the actual inventor after conception of the claimed invention" *Ethicon*, 135 F.3d, at 1460 (citations omitted). The law is clear that such persons are not inventors. *See Burroughs Wellcome*, 40 F.3d at 1228 ("[T]he discovery that an invention actually works is part of its reduction to practice"); *Ethicon,* 135 F.3d at 1460 (one does not become a joint inventor "simply reduc[ing] the inventor's idea to practice.").

133. Not a single witness, including Dr. Costall, claimed that she or her colleagues were inventors of the claimed subject matter. "When an alleged omitted co-inventor does not claim to be such, it can hardly be inequitable conduct not to identify that person to the PTO as an inventor." *Pro-Mold & Tool Co. v. Great Lakes Plastics, Inc.*, 75 F.3d 1568, 1576 (Fed. Cir. 1996).

134. Section 102(f) is irrelevant to the issues in this case. Because Dr. Owen and Dr. Costall worked together following Dr. Owen's conception of the invention, the work of Dr.

Costall at best raises an issue of inventorship, not "derivation" under § 102(f). If Dr. Costall contributed to a complete conception of the invention, she might be a "joint inventor," and the appropriate remedy would be to correct inventorship under 35 U.S.C. § 256. Dr. Costall's contribution, however, does not rise to the level of co-inventorship because her work occurred after Dr. Owen's conception and merely confirmed his prior invention. *See Burroughs Wellcome*, 40 F.3d at 1228.

135. Teva's contention that one of Dr. Owen's laboratory technicians, Annette Wright should have been an inventor or co-inventor of the subject matter claimed in the '860 patent is both waived and meritless.

136. Teva did not raise its contention that Ms. Wright should be a co-inventor at any time until its *post*-trial briefing. Ms. Wright was disclosed as a knowledgeable witness early in the discovery period, yet Teva never sought her deposition during fact discovery. *See* paragraphs 8, 83, *supra*. Teva did not plead with particularity any claim that Ms. Wright was fraudulently omitted as an inventor as required under Fed. R. Civ. P. 9(b), *see Ferguson Beauregard/Logic Controls, Division of Dover Resources, Inc. v. Mega Systems, LLC*, 350 F.3d 1327, 1344 (Fed. Cir. 2003); *see also EMC Corp. v. Storage Technologies Corp.*, 921 F. Supp. 1261, 1263 (D. Del. 1996)("the particularity requirement of Rule 9(b) applies to inequitable conduct charges"), nor did it identify these contentions in pre-trial filings as required by this Court's local rules. D. Del. Civ. Proc. & Practice § 16.4(d) (1995). Accordingly, Teva cannot now assert such a claim.

137. Furthermore, there is no evidence, much less clear and convincing evidence, that Dr. Owen's lab technician, Annette Wright, either alone or jointly with anyone else, conceived the idea of using ropinirole to treat Parkinson's Disease. The record evidence shows that Ms. Wright merely conducted cardiovascular experiments under Dr. Owen's direction that led to

behavioral observations from which Dr. Owen concluded that ropinirole had anti-Parkinson's potential. *See* paragraphs 23-28, 83, *supra*. Performing the experiments on which Dr. Owen's conclusions were based does not make her an inventor. *See, e.g., Acromed Corp. v. Sofamor Danek Group, Inc.*, 253 F.3d 1371, 1380-81 (Fed. Cir. 2001) (rejecting machinist's claim to joint inventorship given that he did no more than follow the actual inventor's instructions, which required only the exercise of the normal skills expected of an ordinary machinist); *see also, Mueller Brass Co.*, 352 F. Supp. at 1373 ("[A] lab technician who carried out a certain experiment under instructions of his superiors, recorded the results, and moved on to other things . . . was not a co-inventor of the claimed method.").

138. Teva has also failed to carry its burden of establishing inequitable conduct with respect to the genus claim (claim 1) in the '860 patent. Teva has failed to identify anyone else besides Dr. Owen who contributed to the invention of this claim. Indeed, Dr. Peter Giddings – the only currently-employed GSK patent attorney who had any role in the prosecution of the application that became the '860 patent – expressly denied that he was an inventor of claim 1. *See* paragraph 54, *supra*. Teva has also provided no evidence that any of the former GSK patent attorneys whose names appear on the '860 patent, including the Honorable Alan Lourie, played a role in the invention and deceptively withheld it from the PTO.

139. Including a genus claim of the type claimed in claim 1 of the '860 patent is consistent with industry practice. *See* paragraphs 50-53, *supra*. Moreover, the uncontradicted testimony of Dr. Bartlett establishes that the scope of the '860 genus claim is reasonable in light of the prior art indolone patents including the '808 patent, which is broader in many respects and which Teva is no longer challenging in this action. *See* paragraphs 56-61, *supra*. In addition, notwithstanding Dr. Long's speculation that a small number of compounds within the '860 genus

would be inactive, there is no record evidence establishing any such compounds are ineffective in treating Parkinson's Disease or that anyone involved in the prosecution of the '860 patent had any reason to believe that was the case during the relevant time period. *See* paragraphs 62-73, *supra*.

140. Finally, Teva has failed to establish by clear and convincing evidence any intent to deceive on GSK's part with respect to the inventorship determination for the '860 patent. Indeed, Teva has failed to identify any motive from which an intent to deceive could be inferred. Because Bradford was under contract to assign any results or intellectual property rights to GSK, there was no motive for GSK to conceal any role Bradford may have played in the invention. *See* paragraphs 35, 84, *supra*. Similarly, there was no motive to conceal the role, if any, of Annette Wright or any GSK patent attorney because, like Dr. Owen, they were employees of the company at the time of the invention. *See, e.g., Modine Mfg. Co. v. Allen Group, Inc.*, 917 F.2d 538, 542 (Fed. Cir. 1990) (upholding finding of no intent to deceive where party had "presented evidence tending to show that any failure to disclose was inadvertent, and that in any case the inventor would have had no motive to intentionally lie about inventorship because the company would own any patent that might (and did) issue.").

B. Alleged Misstatements in Patent Specification

141. Teva has also failed to carry its burden of proving by clear and convincing evidence that GSK made any material misstatements or omissions in the '860 patent specification with the intent to deceive the PTO.

142. First, the statements in the patent specification about bromocriptine's post-synaptic effects and side-effects were indisputably true. *See* paragraphs 98-109, *supra*. Similarly, the statement about the post-synaptic effects of ropinirole and the other claim

compounds were also true, as reflected by the testimony of Dr. Owen and both parties' pharmacology experts. *See* paragraphs 89-96, *supra*.

143. Second, none of the statements at issue was material to the patentability of the invention claimed in the '860 patent. *See* paragraphs 97, 105-109, *supra*.

144. Third, there is absolutely no evidence of any intent by any of GSK's representatives to deceive the patent office with respect to any of these statements.

C. Relief Requested

145. Teva has failed to carry its burden of proving by clear and convincing evidence that Dr. Owen, Honorable Alan Lourie, Stuart Suter, Vincent Fabiano, or anyone else associated with the '860 patent committed inequitable conduct. Accordingly, there is no basis for finding the '860 patent unenforceable.

146. For the foregoing reasons, GSK is entitled to the following relief:

(a) A declaration that the '860 patent is not invalid or unenforceable;[27/]

(b) A declaration that the submission of Teva's ANDA infringes Claim 3 of the '860 patent;

(c) An order providing that the effective date of any approval of Teva's ANDA shall be a date which is not earlier than the expiration of the '860 patent;

(d) An order permanently enjoining Teva and its affiliates and subsidiaries, and each of their officers, agents, servants, and employees from the commercial manufacture, use, offer to sell, or sale within the United States or importation into

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[27/] This assumes an outcome favorable to GSK on the issue of validity, which is the subject of separate briefing.

the United States of the products that are the subject of Teva's ANDA and from inducing such conduct by others, until after the expiration of the '860 patent; and

(e) Such further and other relief as this Court deems proper and just.

Respectfully submitted,

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CERTIFICATE OF SERVICE

I, Patricia Smink Rogowski, hereby certify that on March 7, 2007 **PLAINTIFFS' PROPOSED FINDINGS OF FACT AND CONCLUSIONS OF LAW REGARDING INEQUITABLE CONDUCT** was filed with the Court Clerk using CM/ECF which will send notification of such filing(s) to Josy W. Ingersoll.

I hereby further certify that on March 7, 2007, I have also served this document on the attorneys of record at the following addresses as indicated:

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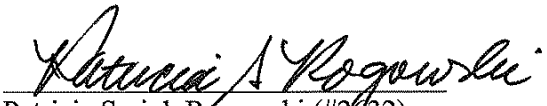

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